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The authors' names should be accompanied by the highest academic or medical degrees that they hold; letters indicating membership in honor or professional societies will not be published. Authors who are in the armed services should not use civilian degrees in connection with their names. The city of each author should be supplied, and an address should be provided at the end of the manuscript for the use of readers who may wish to communicate with the senior author. If academic, hospital, or industrial connections are given for one author, they must be given for all other authors who have such connections. The authors are responsible for obtaining permission from the proper authorities to use the names of institutions.

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Charts and drawings should be in black ink on hard, white paper. Lettering should be large enough to permit necessary reduction. Oversized illustrations should be photographed and a glossy print submitted. Prints of a bluish tinge will not reproduce well. Paper clips must not be used on prints, since the marks left will show in the reproduction. A label should be pasted to the back of each illustration showing its number, the author's name, an abbreviated title of the article, and an indication of the top of the picture. Each chart and illustration must have a descriptive legend, and each table must have a caption.

Journal of Occupational Medicine

Pneumoconiosis Study in Central Pennsylvania Coal Mines

I. MEDICAL PHASE

Jan Lieben, M.D., Harrisburg, Pa.; Eugene Pendergrass, M.D., Philadelphia, Pa.; and W. Wayne McBride, M.D., Harrisburg, Pa.

THE concept that there is little or no pneumoconiosis problem in the bituminous coal industry has prevailed throughout the United States for many years.

The pathological studies of Hart,[1] Gough,[2] Heppleston,[3] Gilson,[4] and the radiologic and epidemiologic studies of Fletcher[5] and his associates have been responsible for a new concept in pneumoconiosis. If Collis[6] could now examine the pathologic and epidemiologic data that have been collected by Gough and his colleagues, we believe that he might modify the statement contained in his scholarly Milroy lecture which reads: "Some dusts such as coal, it is true, not only appear to have no power of producing pneumoconiosis, but even may possess some inhibitory influence on phthisis."

Reorientation concerning the role of dust composition in the pneumoconiosis problem of the bituminous coal workers has been difficult. For many years it was widely believed that disability was the result of the fibrogenic effects of finely divided silica only and that coal dust was definitely harmless.[7]

Dr. Lieben is Director of the Division of Occupational Health, Pennsylvania Department of Health. Dr. Pendergrass is Professor of Radiology, University of Pennsylvania School of Medicine, and Director, Department of Radiology, University Hospital. Dr. McBride is a public health research physician, Division of Occupational Health, Pennsylvania Department of Health.

Presented in part at the Forty-Sixth Annual Meeting of the Industrial Medical Association, Los Angeles, Calif., April 11, 1961.

Recently cases of pneumoconiosis have been reported among coal trimmers in Great Britain whose only exposure consisted of loading coal into ships. Other cases have been reported in persons who have been exposed to graphite, which is pure carbon with only traces of free silica.[1, 4, 8] Nevertheless, there are those who believe that the small quantities of free silica which are found with coal or graphite may be the causative agent of pneumoconiosis.[7]

The authors feel that there are few miners in the United States who have been exposed to coal dust only. The silica content of airborne dust in the mines surveyed in the present study varied with the operation, from 1% during coal loading to 48% during rock drilling (Table VIII, Baier and Diakun, this issue[15]).

Clarke and Moffet,[21] some 20 years ago, reported a study of underground workers at a bituminous mine in the Southern Appalachian coal field. It was found that about 1% of the underground workers had silicosis, that cases occurred among coal loaders after 20 years' employment at this job, and that undercutting machine operators developed the condition in a shorter time. Beyond this report, there has been relatively little information available on the effects of the working environment on the coal miner in the United States. We have lacked the good epidemiologic studies needed to define the problem.

In view of these unanswered questions, the Division of Occupational Health of the Pennsylvania Department of Health initiated a sur-

vey among the membership of several local unions in the Pennsylvania bituminous coal areas. As a result, it was possible to organize the first phase of the present study, in which 5,072 coal workers in central Pennsylvania were examined between April and November, 1959. Simultaneously, fourteen mines of the area were studied by the Division's industrial hygienists. Their findings on the working environment, the dust counts, and the geology of the area are described in a companion paper by Baier and Diakun.[15]

The Survey

The survey included a short personal and occupational history for individuals examined, a 14 × 17 inch chest roentgenogram, vitalometer readings, and five standard British questions on subjective degree of shortness of breath.

The x-ray examinations were conducted by a mobile team which operated at 14 different locations. Working, unemployed and retired miners, foremen, and other salaried mine employees participated in the study.

The majority of those surveyed came from three coal-producing counties of central Pennsylvania. The total mining population of the area was approximately 20,000, including an undetermined number of pensioners.

The history form, reproduced in Figs. 1a and 1b, was either prepared by the miner and reviewed by a staff member or completed by a member of the surveying team at the time of the interview. The form constitutes a compromise between the information which is desired and the amount of information which can reasonably be obtained from a miner who presents himself on his own time, before or after a full day's work. A history of smoking was omitted for this reason.

The Gaensler modification of the electronically timed Collins vitalometer was used for the three-second and total vital capacity.[21] The 14 × 17 inch roentgenograms were taken by registered x-ray technicians with a 200 ma x-ray machine mounted in a bus. This machine was equipped with a voltage regulator, synchronous time switch, and full wave valve tube rectification. The rotating anode tube target was 1.5 × 1.5 mm. The minimum kilovoltage

HIH-19024

COMMONWEALTH OF PENNSYLVANIA
DEPARTMENT OF HEALTH
OCCUPATIONAL HEALTH

SILICOSIS STUDY QUESTIONNAIRE

1. UNION LOCAL | 2. REG. AND X-RAY NO.

3. NAME
LAST FIRST MIDDLE

4. ADDRESS
STREET CITY STATE

5A. AGE | 5B. SEX M ☐ F ☐ | 5C. RACE W ☐ NON-W ☐

5D. PHYSICIAN TO WHOM REPORT SHOULD BE SENT IF NECESSARY:
NAME | ADDRESS

MEDICAL HISTORY

6. HAVE YOU EVER HAD:			AT WHAT AGE
A. TUBERCULOSIS	YES ☐	NO ☐	
B. PNEUMONIA	YES ☐	NO ☐	
C. PLEURISY	YES ☐	NO ☐	
D. HEART TROUBLE	YES ☐	NO ☐	
E. BRONCHIAL ASTHMA	YES ☐	NO ☐	
F. HAY FEVER OR OTHER ALLERGIC CONDITIONS	YES ☐	NO ☐	

7. HOW MANY COLDS DO YOU GET EACH YEAR?

8. OTHER DISEASES?

9. DO YOU USUALLY HAVE A COUGH?			
	A. YES ☐	NO ☐	
IF YES: AT NIGHT?	B. YES ☐	NO ☐	
IN DAY-TIME?	C. YES ☐	NO ☐	
ON ARISING?	D. YES ☐	NO ☐	

10. DO YOU RAISE MUCUS (PHLEGM)?	A. YES ☐	NO ☐
IF YES, HOW MUCH EACH DAY?	B. LESS THAN 1 TABLESPOON ☐	
	C. 1 TABLESPOON OR MORE ☐	
	D. A CUP FULL ☐	

11. ARE YOU TROUBLED BY SHORTNESS OF BREATH?	YES ☐	NO ☐
A. IF NO: NOT EVEN ON HURRYING ON THE LEVEL OR WALKING UP A SLIGHT HILL?	YES ☐	NO ☐
B. IF YES: DO YOU WALK SLOWER THAN PEOPLE YOUR OWN AGE ON THE LEVEL DUE TO SHORTNESS OF BREATH?	YES ☐	NO ☐
C. IF YES: DO YOU EVER HAVE TO STOP FOR BREATH WHEN WALKING AT YOUR OWN PACE ON THE LEVEL?	YES ☐	NO ☐
D. IF YES: DO YOU HAVE TO STOP FOR BREATH AFTER WALKING ABOUT TWO BLOCKS ON THE LEVEL GROUND?	YES ☐	NO ☐
E. IF YES: ARE YOU SHORT OF BREATH WHEN DRESSING OR UNDRESSING?	YES ☐	NO ☐

12. DO YOU GET PAIN IN THE CHEST?	YES ☐	NO ☐
A. IF YES: WHERE		
B. IF YES: IS IT RELATED TO ACTIVITY?	YES ☐	NO ☐
C. IF YES: IS IT RELIEVED BY REST?	YES ☐	NO ☐
D. IF YES: DOES THE PAIN MOVE?	YES ☐	NO ☐

OCCUPATIONAL HISTORY

13A. ARE YOU NOW LAID OFF?	YES ☐	NO ☐	13B. IF YES, SINCE WHEN		
14A. ARE YOU RETIRED?	YES ☐	NO ☐	14B. IF YES, SINCE WHEN		
IF NO, IS YOUR PRESENT JOB:					
14C. UNDERGROUND: FULL-TIME?	YES ☐	NO ☐	14D. PART-TIME?	YES ☐	NO
14E. SURFACE: FULL-TIME?	YES ☐	NO ☐	14F. PART-TIME?	YES ☐	NO

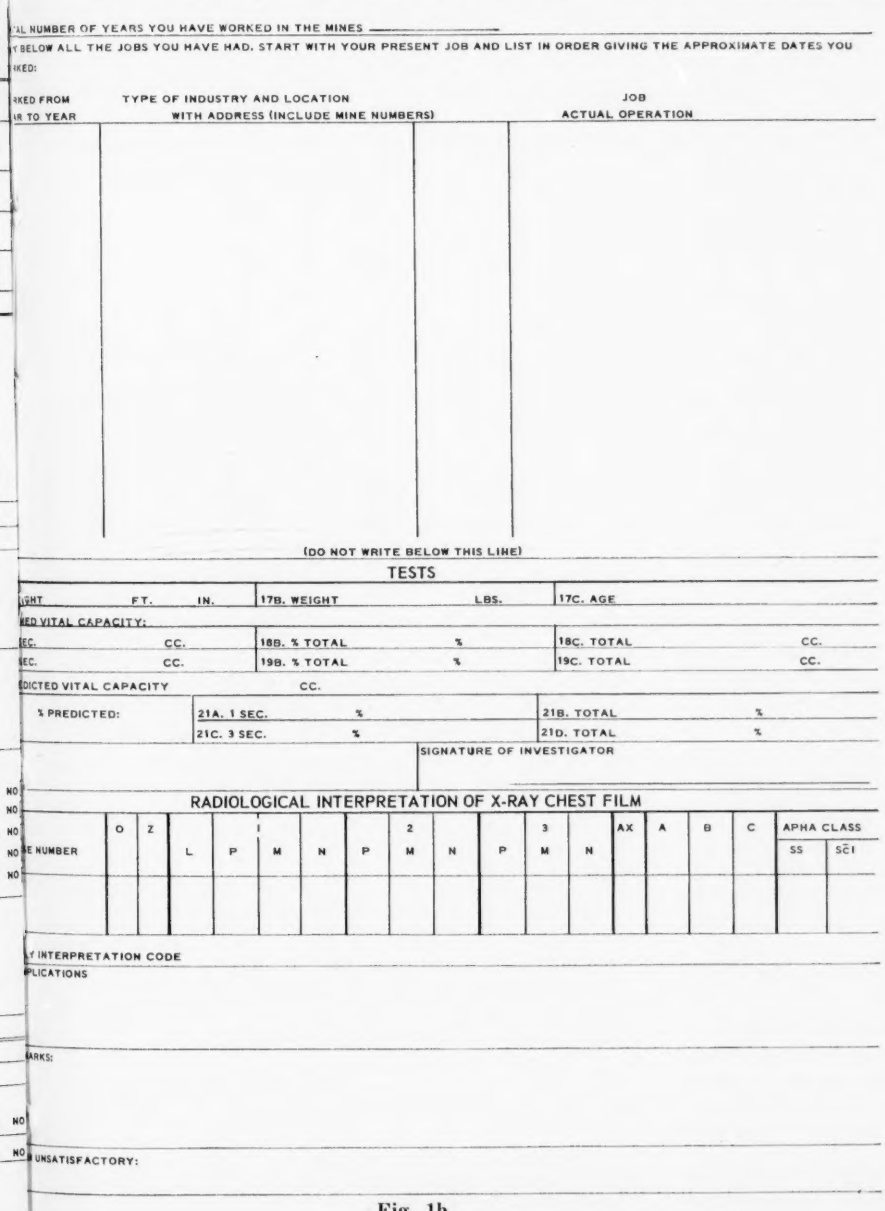
Fig. 1a

was 70, and the average exposure time 0.1 second. The maximum voltage drop was 10 volts.

The tube-film distance was fixed at 6 feet. A general purpose medium sensitivity film was utilized throughout the study. Cassettes were equipped with medium speed intensifying screens. Electric current was provided in each instance by directly tapping the transformer or the power lines.

All the films were developed automatically and read by the same team of readers of the University of Pennsylvania Hospital, headed by one of us (E. P.). At the time the films were read the radiologists did not have the examinees' age, history, or vital capacity results available.

The three-second timed vital capacity, utilizing the modification of Gaensler, was measured for each individual. This modification was selected because its simplicity, ease of administration, and small observer error made it ideal for field screening, and results have shown a favorable comparison with more detailed laboratory studies.[9] In three seconds 95% or more of the total vital capacity measurement is achieved. With a normal rate of 15 or more respirations per minute, one second is required for inspiration and three seconds for expiration: therefore three seconds is a functional value.[10] The one-second time interval was not used because accuracy in measuring so short an interval is difficult to obtain.[11] The regression formula based on age, sex, and height was used in calculating the predicted vital capacity.[12] In keeping with the need to hold the survey to a set of screening procedures, the examinees were required to make usually one expiratory effort for this measurement, but on occasion two or three were necessary.


AL NUMBER OF YEARS YOU HAVE WORKED IN THE MINES

Y BELOW ALL THE JOBS YOU HAVE HAD. START WITH YOUR PRESENT JOB AND LIST IN ORDER GIVING THE APPROXIMATE DATES YOU

KED:

KED FROM TO YEAR | TYPE OF INDUSTRY AND LOCATION WITH ADDRESS (INCLUDE MINE NUMBERS) | JOB ACTUAL OPERATION

(DO NOT WRITE BELOW THIS LINE)

TESTS

IGHT FT. IN. | 17B. WEIGHT LBS. | 17C. AGE

ED VITAL CAPACITY:

EC. CC. | 18B. % TOTAL % | 18C. TOTAL CC.

EC. CC. | 19B. % TOTAL % | 19C. TOTAL CC.

DICTED VITAL CAPACITY CC.

% PREDICTED: | 21A. 1 SEC. % | 21B. TOTAL %

21C. 3 SEC. % | 21D. TOTAL %

SIGNATURE OF INVESTIGATOR

RADIOLOGICAL INTERPRETATION OF X-RAY CHEST FILM

E NUMBER	0	Z	1 L	1 P	1 M	1 N	2 P	2 M	2 N	3 P	3 M	3 N	AX	A	B	C	APHA CLASS SS	APHA CLASS SCI

Y INTERPRETATION CODE

PLICATIONS

ARKS:

UNSATISFACTORY:

Fig. 1b

Roentgenographic Classification

All films were classified according to the "International Classification of Persistent Radiological Opacities in the Lung Fields Provoked by the Inhalation of Mineral Dusts," from the "Classification of Radiographs of the Pneumoconioses," published by the International Labor Office in 1958. It was intended that this definition should cover all coal and carbon dusts.

For the pneumoconiosis classification of coal miners' roentgenograms small and large opacities are recognized.

Table I—Working Miners—Age and Roentgenographic Lung Findings

International Classification of Pneumoconiosis	Age						
	20-34	35-44	45-54	55-64	65 and Over	Age Unknown	Total
No pneumoconiosis	390 (95%)	1,194 (81%)	977 (61%)	358 (53%)	16 (57%)	2	2,937
Pneumoconiosis							
Suspected	8	58	102	44	2	....	214
Category 1	2	35	64	36	1	....	138
Category 2	8	79	110	56	....	....	253
Category 3	1	37	80	25	....	....	143
Coalescent lesions							
Suspected	....	32	69	36	1	2	140
Category A	....	13	60	36	3	....	112
Category B	....	26	116	80	4	....	226
Category C	....	....	9	3	1	....	13
Film unsatisfactory	....	....	3	3	....	....	6
Totals	409	1,474	1,590	677	28	4	4,182

Depending on the size of the small opacities, three types appear in the classification:

p = predominance of punctiform opacities up to 1.5 mm in diameter

m = predominance of micronodular opacities 1.5 to 3 mm in diameter

n = predominance of nodular opacities 3 to 10 mm in diameter

The number of small opacities are classified in three categories:

Category 1 = a small number of opacities in an area equivalent to at least two anterior rib spaces and not greater than one-third of the lung field.

Category 2 = opacities more numerous and diffuse than in category 1 distributed over most of the lung field

Category 3 = very numerous profuse opacities covering the whole or very nearly the whole of the lung field

Cases in which coalescence was suspected but not definite as in category A below were designated as AX.

The size and number of large opacities are divided into three categories:

Category A = an opacity having the longest diameter of between 1 and 5 cm or several opacities each greater than 1 centimeter, the sum of whose longest diameter does not exceed 5 cm

Category B = one or more opacities larger or more numerous than those in category A whose combined area does not exceed half of the lung field

Category C = one or more large opacities whose combined area exceeds half of the lung field

Table II—Retired Miners—Age and Roentgenographic Lung Findings

International Classification of Pneumoconiosis	Age			
	20-54	55-64	65 and Over	Total
No pneumoconiosis	25 (63%)	127 (54%)	204 (39%)	356
Pneumoconiosis				
Suspected	2	13	33	48
Category 1	2	10	24	36
Category 2	2	15	40	57
Category 3	3	11	35	49
Coalescent lesions				
Suspected	1	15	37	53
Category A	....	10	36	46
Category B	5	27	100	132
Category C	....	4	12	16
Film unsatisfactory	....	1	2	3
Totals	40	233	523	796

Roentgenographic Findings

Tables I through IV* present the age distribution and the length of exposure in the working environment for both working and retired miners. It is interesting to note from Table I that the percentages of those with "no pneumoconiosis" decreases as the age progresses, from 95% in the 20 to 34 year age group to 53% in the 55 to 64 year age group. This trend continues in Table II, where only 39% are found with "no pneumoconiosis" in the group 65 years of age and over. A similar

*The data of 94 cases have been omitted from Tables I-IV because records were not clear as to present employment or retirement.

Table III—Working Miners—Years Worked and Roentgenographic Lung Findings

International Classification of Pneumoconiosis	Number of Years Worked						
	Under 20	20-24	25-29	30-39	40 and Over	Unknown	Total
No pneumoconiosis	1,058 (91%)	556 (76%)	421 (67%)	671 (54%)	187 (52%)	44 (76%)	2,937
Pneumoconiosis							
Suspected	34	33	34	82	27	4	214
Category 1	16	16	26	61	16	3	138
Category 2	29	52	42	104	26	....	253
Category 3	10	24	23	71	15	....	143
Coalescent lesions							
Suspected	8	20	32	58	19	3	140
Category A	6	12	20	51	22	1	112
Category B	7	21	33	121	41	3	226
Category C	....	....	....	11	2	....	13
Film unsatisfactory	....	....	1	5	....	....	6
Totals	1,168	734	632	1,235	355	58	4,182

trend is found when the length of time worked in the mines and the number of men with no pneumoconiosis are analyzed (Tables III and IV). Of the working men with less than 20 years in the mines 91% have no roentgenographic evidence of pneumoconiosis. This number gradually decreases to 52% of those who have worked 40 years or more. This decrease continues in the retired group (Table IV). Only 40% of the retired group who have worked 40 years or more were found to be without evidence of pneumoconiosis.

Table IV—Retired Miners—Years Worked and Roentgenographic Lung Findings

International Classification of Pneumoconiosis	Number of Years Worked				
	Under 30	30-39	40 and Over	Years Unknown	Total
No pneumoconiosis	46 (69%)	108 (48%)	196 (40%)	6 (40%)	356
Pneumoconiosis					
Suspected	4	8	36	....	48
Category 1	2	11	23	....	36
Category 2	1	16	40	....	57
Category 3	....	12	36	1	49
Coalescent lesions					
Suspected	3	13	37	....	53
Category A	4	13	27	2	46
Category B	7	38	82	5	132
Category C	....	4	11	1	16
Film unsatisfactory	....	1	2	....	3
Totals	67	224	490	15	796

Of the working miners, 29% either have some stage of pneumoconiosis or are suspected of having the disease. This finding is undoubtedly influenced by the depressed employment in the industry at the time of the study and a seniority policy that protects the job security of older workers. Because of these factors, the age group expected to have the highest number of men without pneumoconiosis —that from 20 to 35 years of age—was small in number. There were only 409 in this group, as opposed to 3,741 in the age group from 35 to 64 years of age. In miners over 35 years of age the exposure, of course, has been of much longer duration.

In this series of roentgenograms of bituminous miners all the types of shadows seen in many hard coal miners who have worked for years in the Pennsylvania anthracite mines were observed (Fig. 2). Emphysema, although present, was not a prominent finding. Opacities of varying types were found. These opacities varied from small micronodular densities to large massive lesions. The nodules were distributed unilaterally or bilaterally in any part of the lung (Figs. 3, 4, and 5). Occasionally, large massive lesions were seen unilaterally or bilaterally without any background of micronodular densities (Fig. 6). The unilateral massive lesions required careful evaluation to diagnose or exclude carcinoma of the lung (Fig. 7).

Evaluation of the significance of so-called prominent vascular markings and their relation to pneumoconiosis was impossible in many instances. For the most part the author of this portion of the manuscript (E. P.) is prone to accept the concept that pneumoconiosis tends to render vascular patterns less prominent.

The roentgen appearances of the lungs in

these soft coal miners were somewhat unexpected. In previous studies of roentgenograms of soft coal miners very few opacities were observed. For the most part, emphysema with or without coalescent lesions was found in the upper lobes.

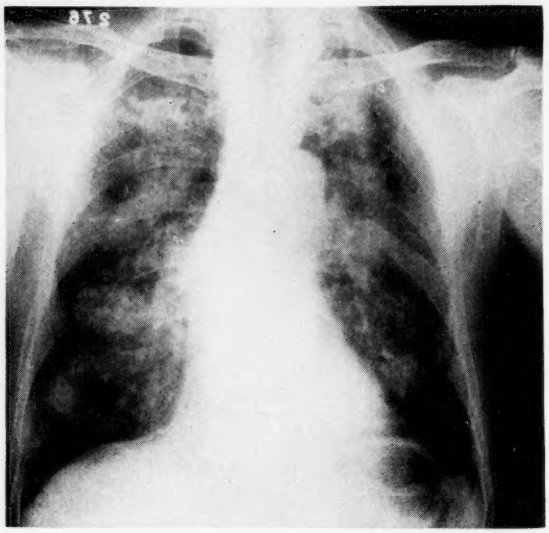


Fig. 2—Extensive changes throughout both lungs in a soft coal miner, aged 67 years. He is retired and has worked underground for 43 years as a laborer and at hand loading. He has symptoms of shortness of breath, a slight productive cough, and marked reduction in vital capacity.
The pattern of abnormal changes are similar to those seen in a hard coal miner. Compare with Fig. 8.

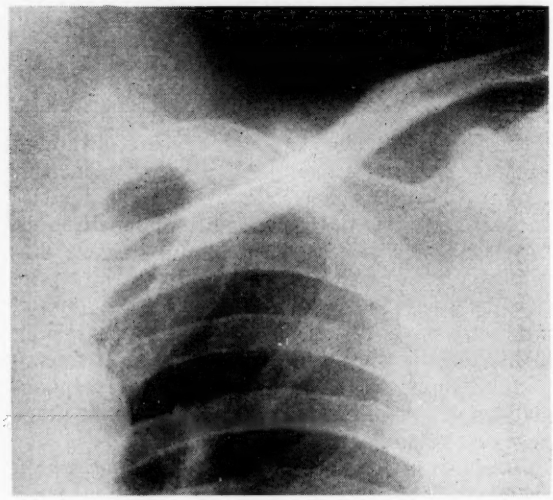


Fig. 3—Unilateral nodulation, 1 m, left upper lobe in a soft coal miner, 71 years old, who loaded coal underground for 41 years (retired 7 years). He has a slight productive cough during the day and is not short of breath. The individual nodules are slightly larger than those seen in Fig. 2. The changes are limited to the left upper lobe.

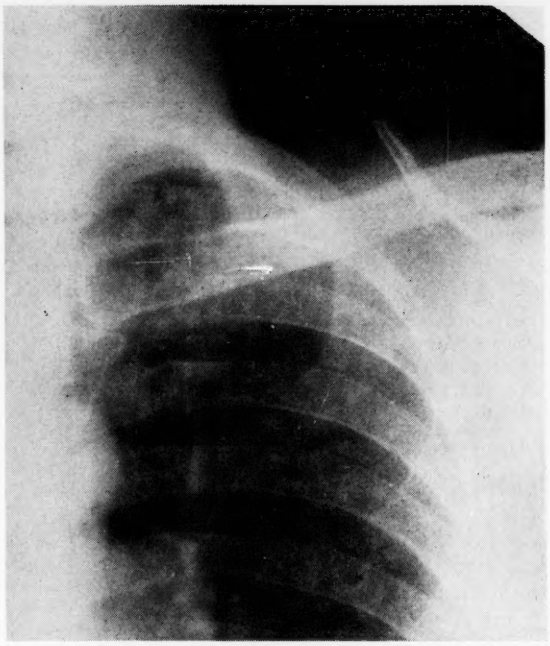


Fig. 4—Bilateral nodulation, 2 n, in a soft coal miner 50 years old. He has no symptoms, and his vital capacity is normal. He has worked underground for 35 years. The nodular pattern is more extensive and the individual nodules are larger than those seen in Figs. 2 and 3.

Another unexpected observation was the presence of abnormal changes in the lower lobes. These changes included micronodular densities, lacelike or reticular shadow patterns (similar to those commonly called Kirley's lines), and coalescent and massive lesions. Some of the shadow patterns simulated those found in moderately advanced asbestosis (Figs. 8 and 9). In some instances, the shadow densities were more dominant in lower lung fields than in the upper, a finding just the opposite of that ordinarily observed in hard coal miners (Figs. 10 and 11). The impression was obtained that there must be some modifying factor to account for the extensive lower lung abnormalities.

The pleural changes for the most part were similar to those seen in anthracosilicosis. However, a significant number of soft coal miners showed evidence of calcification of the pleura. The extent of calcification varied from small plaques over the domes of the diaphragm and the lateral chest wall to extensive plaques. The calcification was either unilateral or bilateral.

The presence of calcified pleura was of interest because an increasing number of reports are being made concerning the presence of calcified pleura in asbestosis. It is well known that the major early changes in asbestosis occur in the lower thorax. These soft coal miners

in many instances seem to show the major changes in the lower lobes. The question can be raised as to whether the extensive lower lobe changes could be related to the increased incidence of calcified pleura.

Shortness of Breath

Each examinee was asked five questions regarding his degree of shortness of breath (Section 11, Fig. 1a). An affirmative answer to questions B, C, D, and E indicated various degrees of shortness of breath, with an affirmative answer to B expressing the mildest and E the severest form. It is interesting to note that of 3,368 examinees with no roentgenographic evidence of pneumoconiosis 1,520, or 45%, answered the question, "Are you troubled by shortness of breath" affirmatively, and 811, or 24%, of the total group gave answers indicating the severe degrees of shortness of breath (Table V). Also of interest is the fact that of 750 examinees with either suspected coalescence or coalescent pneumoconiosis on their roentgenographic films 244, or 32.5%, disclaimed any shortness of breath.

Table V supports the conclusion reported by British and other observers that there is poor correlation between the roentgenographic findings and the degree of shortness of breath. The 244 examinees with suspected coalescence or coalescent lesions and no shortness of breath,

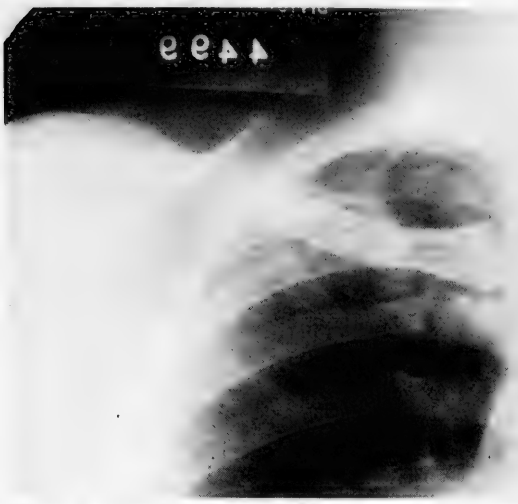


Fig. 5—Unilateral nodulation, 1 m, right upper lobe, in a soft coal miner, 51 years of age, who works underground and has done so for the past 35 years (loader). He says he has a daily productive cough (15 cc) and is short of breath on walking up a slight hill. The timed vital capacity is 500 cc. below his predicted normal. The pattern of nodulation is confined to the right upper lobe. This is seen in a number of instances on either the right or the left side, without abnormal shadows elsewhere.

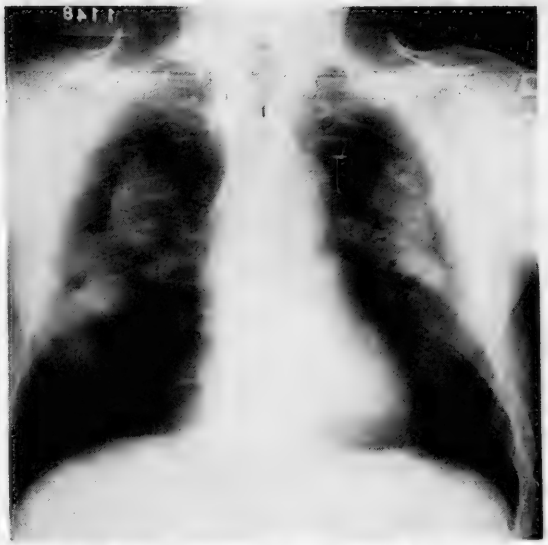


Fig. 6—Massive lesions in each lung of a soft coal miner, 63 years of age. He is retired and has worked 25 years underground as a driller and loader. He has a productive cough (30 cc), is markedly short of breath, and has pain in the left chest. His vital capacity is reduced 75%.
Note that one does not see a pattern of nodulation—only massive lesions.

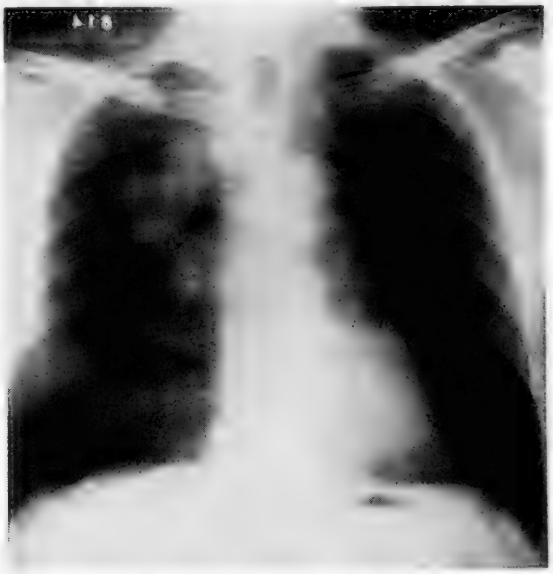


Fig. 7—Unilateral massive lesion in a 73-year-old soft coal miner with a slight degree of nodulation in the left lung. He is retired but worked underground for 41 years, shooting rock and loading coal. He has a productive cough (1 cup), shortness of breath, pain in the stomach (ulcer), arthritis of the spine, and 30% of the predicted vital capacity.
The massive lesion is often diagnosed carcinoma. Complete studies are necessary to identify the nature of the lesion, silicotic mass or carcinoma. Sometimes operation is the way to make a definite diagnosis.

as well as the 811 persons with severe shortness of breath and no pneumoconiosis, invite further studies.

In comparing age with the degree of shortness of breath in Fig. 12 it is noted that of 422 miners under the age of 35 years, 23.2% have this complaint. Of this number, 8.1% indicated the severest degree of shortness of breath. Of 553 miners over 65 years of age, 73.8% gave affirmative answers to the question on shortness

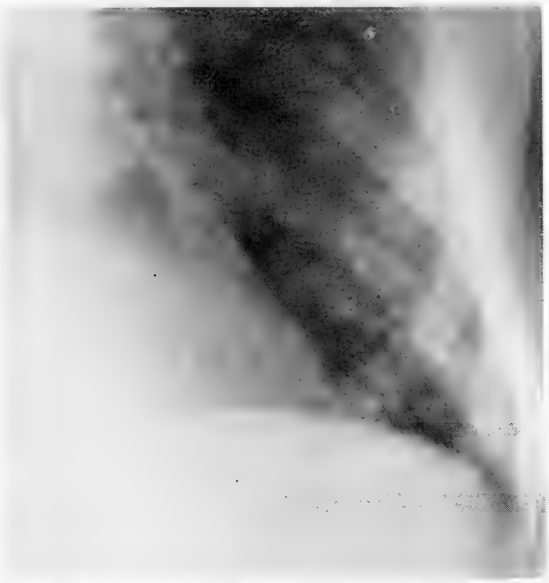


Fig. 8—Pattern of nodulation throughout both lungs in a 71-year-old soft coal miner, who is without symptoms except a slight shortness of breath when he walks up a slight hill. He has worked underground for 41 years.
The lesions (nodules) are distributed throughout both lungs, but the lower lobes are more involved than the other portions of the lung.

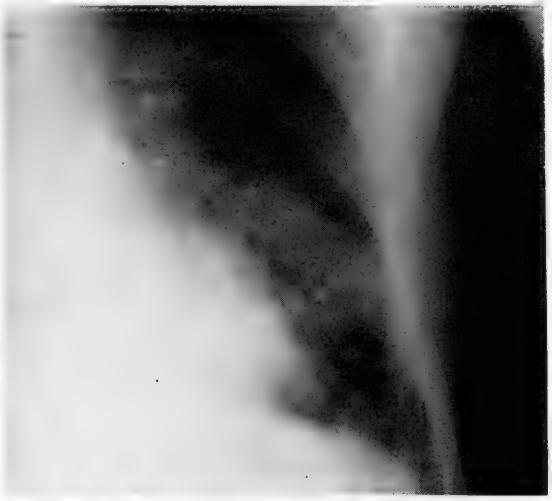


Fig. 9—Micronodular pattern and conglomeration 2p, A, in a 69-year-old soft coal miner who is without symptoms but has reduction in his vital capacity. He has worked 45 years underground as a loader.
The abnormal pattern is limited to the lower lobes, especially on the left side. The individual nodules are so small that they are difficult to distinguish.

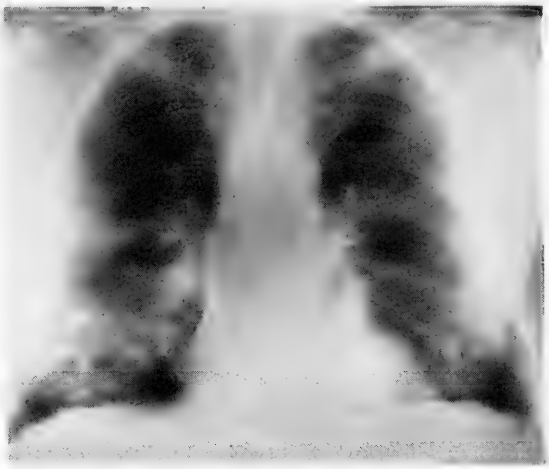


Fig. 10—Nodular and conglomerate pattern of silicosis in a 59-year-old soft coal miner who has worked for 46 years loading coal underground. He has a productive cough, is short of breath, and has a marked reduction in vital capacity.
Note that the changes are confined to the lower two-thirds of the lung fields and are more pronounced on the right side.

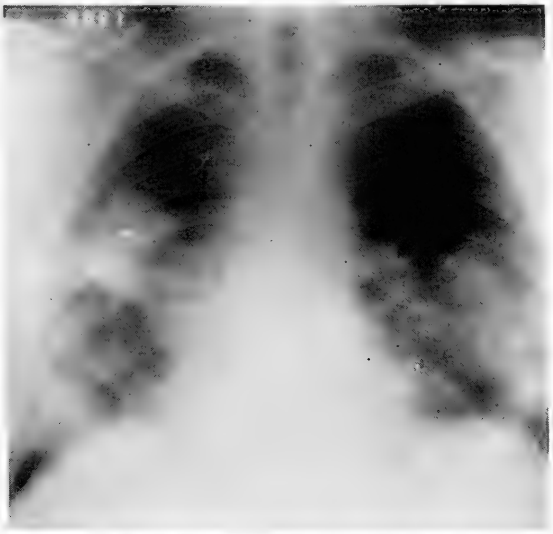


Fig. 11—Massive conglomerate lesions in a 61-year-old soft coal miner. He has mined coal for 46 years and has worked full time underground. He has cough but very little expectoration. He has pain in his chest and is short of breath. His vital capacity is about 1,000 cc below the predicted normal.
Note that there is very little evidence of a nodular background, most of the lesions being massive and confined to the lower two-thirds of the chest.

Table V—Shortness of Breath and Roentgenographic Lung Findings

International Classification of Pneumoconiosis	Shortness of Breath: No — Symptom Disclaimed	No — Degree A*	No — Subtotal		Yes — Degrees B C D E*	Yes — Degree not Stated	Yes — Subtotal	Total
No pneumoconiosis	1,437	411	1,848		811 (24%)	709	1,520 (45%)	3,368
Pneumoconiosis								
Suspected	98	23	121		90	53	143	264
Category 1	70	18	88		52	36	88	176
Category 2	109	46	155		96	61	157	312
Category 3	61	23	84		75	34	109	193
Coalescent lesions								
Suspected	57	27	84		72	41	113	197
Category A	42	19	61	(32.5%)	56	42	98	159
Category B	70	27	97		197	70	267	364
Category C	2	....	2		23	5	28	30
Film unsatisfactory	2	1	3		4	2	6	9
Total	1,948	595	2,543		1,476	1,053	2,529	5,072

*Degree of shortness of breath
A = short of breath when walking up slight hill (considered as no shortness of breath in this study)
B = short of breath when walking on level with people of own age
C = short of breath when walking at own pace on level
D = short of breath after walking two blocks on level ground
E = short of breath when dressing or undressing

of breath. Included in this group were 328 who were in distress with moderate exertion.

Fig. 13, comparing the degree of shortness of breath with the number of years worked in the mines, shows essentially the same results. A total of 1,216 men interviewed had less than 20 years of work in the mines. Of these, 27.3% stated that they were short of breath. In this group, 122, or 10.0%, indicated the severest degree of shortness of breath. In 853 miners with 40 or more years of service, 70.8% were short of breath, with 446, or 52.3%, indicating shortness of breath of the severest degree.

Fig. 14 shows age and prevalence of cough. Of the 422 miners under the age of 35 years, 27.7% answered that they had a cough. The prevalence of cough progressively increases

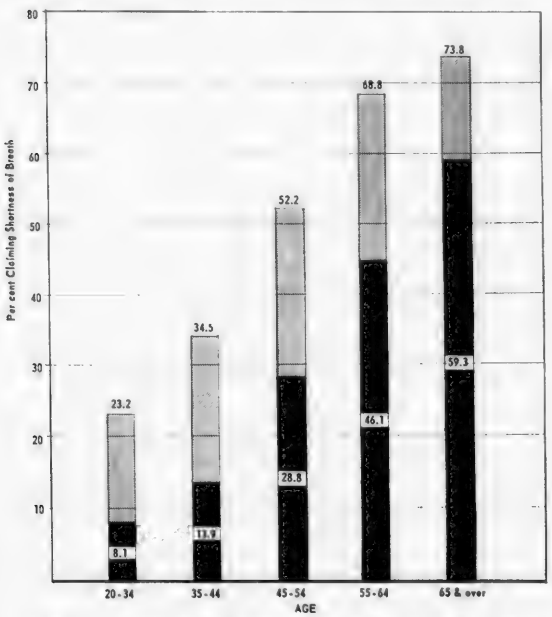


Fig. 12—Degree of shortness of breath by age groups. Severest degree of shortness of breath is indicated by solid black.

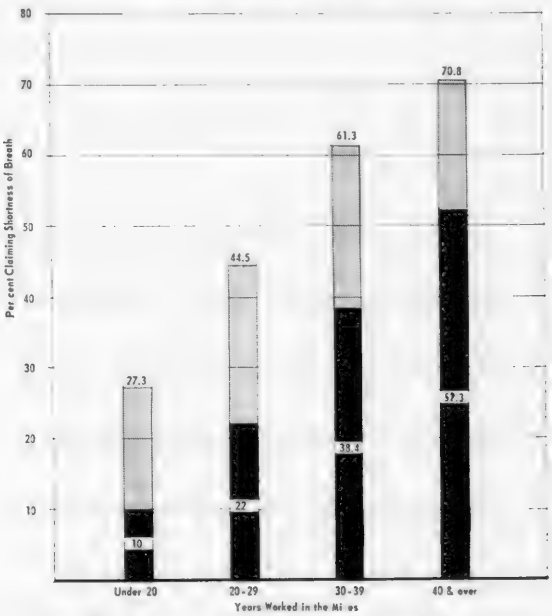


Fig. 13—Degree of shortness of breath by years worked in the mines. Severest degree of shortness of breath is indicated by solid black.

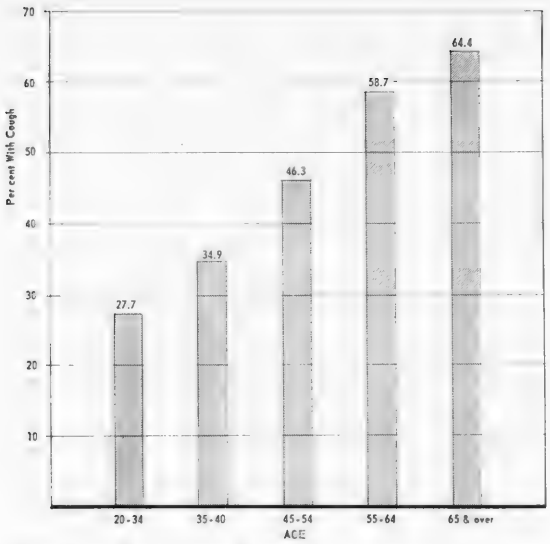


Fig. 14—Prevalence of cough by age groups.

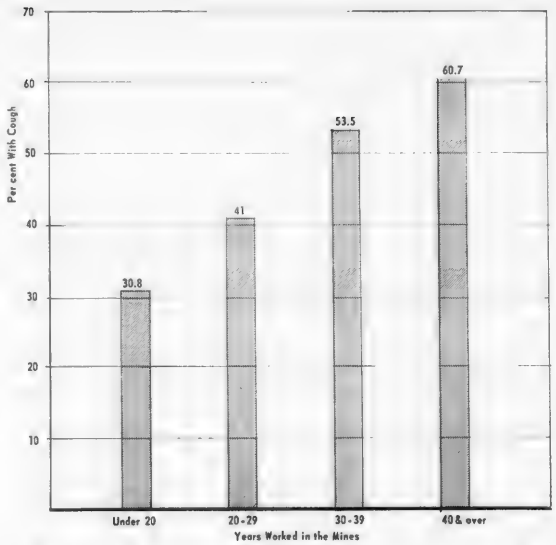


Fig. 15—Prevalence of cough by years worked in the mines.

with age, reaching a peak of 64.4% of 553 men in the age group of 65 years of age and above.

Fig. 15 shows approximately the same findings when the prevalence of cough and the number of years in the mines are tabulated. Of the 1,216 miners with less than 20 years of service in the mines, 30.8% reported cough. This increases to a prevalence of 60.7% with cough of a total of 853 men with 40 or more years of work in the mines.

Table VI shows the prevalence of cough with the classification of pneumoconiosis. An interesting observation is that of 3,368 miners with no pneumoconiosis 42.3% reported cough. Of a total of 750 miners with coalescent or suspected coalescent pneumoconiosis, 57.2% had a cough, and this figure was as high as 76.7% for those with Category C lesions.

Table VII shows the percent of the predicted vital capacity and age distributions. Of the men under 55 years of age, approximately half had predicted vital capacities of 90% and over. Above the age of 55 years, more than one third

Table VI—Cough and Roentgenographic Lung Findings

International Classification of Pneumoconiosis	Cough: Yes			Cough: No		Cough: Unspecified		Total	
No pneumoconiosis	1,424	(42.3%)		1,938	(57.5%)	6	(0.2%)	3,368	
Pneumoconiosis									
Suspected	121			143		....		264	
Category 1	81	(46%)		95		0		176	
Category 2	147	(47.1%)	(48.7%)	164	(51.1%)	1	(0.1%)	312	681
Category 3	104	(53.9%)		89		0		193	
Coalescent lesions									
Suspected	104	(53.3%)		93		0		197	
Category A	85	(53.5%)		73		1		159	
Category B	217	(59.6%)	(57.2%)	146	(42.5%)	1	(0.3%)	364	750
Category C	23	(76.7%)		7		0		30	
Film unsatisfactory	6			3		....		9	
Total	2,312			2,751		9		5,072	

Table VII—All Examined Miners—Vital Capacity and Age

Age Groups	Percentage of Predicted 3-Second Vital Capacity: 90% and Over	70-89%	50-69%	Under 50%	Unknown	Total
Under 45	1,091 (56.2%)	472 (24.3%)	129 (6.6%)	110 (5.7%)	141 (7.3%)	1,943
45-54	704 (42.8%)	517 (31.4%)	200 (12.1%)	125 (7.6%)	99 (6.0%)	1,645
55-64	284 (30.7%)	304 (32.8%)	165 (17.8%)	115 (12.4%)	59 (6.4%)	927
65 and over	107 (19.4%)	147 (26.6%)	140 (25.3%)	118 (21.3%)	41 (7.4%)	553
Unknown	....	....	....	....	4 (100.0%)	4
Total	2,186	1,440	634	468	344	5,072

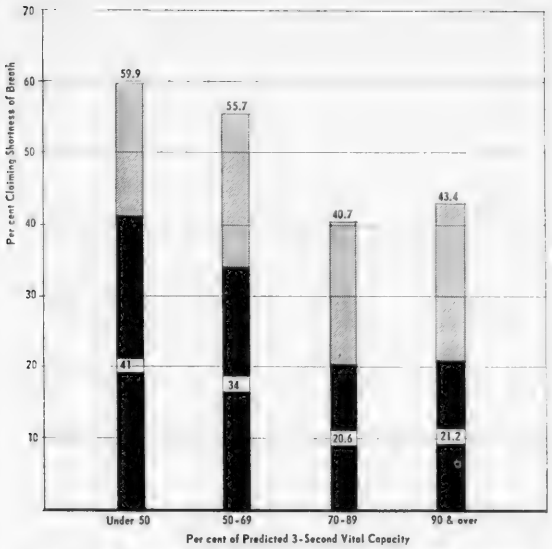


Fig. 16—Degree of shortness of breath and degree of impairment of vital capacity. Severest degree of shortness of breath is indicated by solid black.

of the men had less than 70% of their predicted vital capacities.

It is interesting to note that only 5.7% of the men under 45 years of age had vital capacities under 50% of that predicted, while 21.3% of those over 64 years of age had vital capacities under 50%.

These over-all figures were divided into those for persons with and those for persons without pneumoconiosis. Table VIII shows the percentage of predicted vital capacity versus age in 3,632 miners with no roentgenographic evidence of pneumoconiosis. Of these, 19.3% had three-second vital capacities less than 70% of the predicted values.

The tabulation of predicted vital capacities and age for miners with pneumoconiosis is presented in Table IX. There were 1,440 men in this group, and 27.9% had vital capacities under 70%, an increase of 8.6% over the group with no evidence of pneumoconiosis.

The percent of predicted vital capacity and the classifications of pneumoconiosis are shown

Table VIII—Miners Without Pneumoconiosis—Vital Capacity and Age

Age Groups	Percentage of Predicted 3-Second Vital Capacity					
	90% and Over	70-89%	50-69%	Under 50%	Unknown	Total
Under 45	984	417	113	89	98	1,701
45-54	490	357	138	72	62	1,119
55-64	174	181	102	65	32	554
65 and over	55	66	64	57	14	256
Unknown	....	....	....	....	2	2
Total	1,703 (46.9%)	1,021 (28.1%)	417 (11.5%)	283 (7.8%)	208 (5.7%)	3,632*

*This number includes 264 men with suspected pneumoconiosis.

Table IX—Miners With Pneumoconiosis—Vital Capacity and Age

Age Groups	Percentage of Predicted 3-Second Vital Capacity					
	90% and Over	70-89%	50-69%	Under 50%	Unknown	Total
Under 45	107	55	16	21	43	242
45-54	214	160	62	53	37	526
55-64	110	123	63	50	27	373
65 and over	52	81	76	61	27	297
Unknown	....	....	....	....	2	2
Total	483 (33.5%)	419 (29.1%)	217 (15.1%)	185 (12.8%)	136 (9.5%)	1,440*

*This number includes 9 men whose films were unsatisfactory for precise classification.

Table X—Vital Capacity and Roentgenographic Lung Findings

International Classification of Pneumoconiosis	Percentage of Predicted 3-Second Vital Capacity						
	90% and Over	70-89%	50-69%	Under 50%	Unknown	Total	
No pneumoconiosis	1,607 (47.7%)	962 (28.6%)	390 (11.6%)	238 (7.1%)	171 (5.1%)	3,368	
Pneumoconiosis							
Suspected	96	59	27	45	37	264	
Category 1	69	47	21	15	24	176	
Category 2	126 (38.0%)	88 (26.7%)	31 (13.1%)	33 (10.9%)	34 (11.3%)	312	681
Category 3	64	47	37	26	19	193	
Coalescent lesions							
Suspected	64	71	21	21	20	197	
Category A	54 (29.5%)	52 (31.3%)	22 (16.7%)	17 (14.7%)	14 (7.9%)	159	750
Category B	102	104	74	62	22	364	
Category C	1	8	8	10	3	30	
Film unsatisfactory	3	2	3	1	0	9	
Total	2,186 (43.1%)	1,440 (28.4%)	634 (12.5%)	468 (9.2%)	344 (6.8%)	5,072	

in Table X. Of 5,072 miners tested, we find that 10.9% of those with smaller lesions (pneumoconiosis categories 1, 2, and 3) and 14.7% of those with coalescent or suspected coalescent lesions had vital capacities under 50% of the predicted value. Comparable values in the 90% and over group were 38.0% and 29.5% respectively.

The affirmative answers to the medical history questions on the questionnaire are shown in Table XI, where we find pneumonia and pleurisy to be the most commonly occurring past illnesses.

Vital capacities of workers with positive past medical histories and roentgenographic complications which might influence vital capacity determinations were compiled.

In the group with no roentgen evidence of pneumoconiosis, 19.3% had three-second capacities below 70% of the predicted value (Table VIII). In the group with a positive past medical history and no roentgen evidence of pneumoconiosis, the comparable figure was 24.6% (Table XII).

The group with no evidence of pneumoconiosis was further divided into those without and those with such roentgenographic complications as pleural abnormalities, emphysema, old tuberculosis, tuberculosis, and suspected tuberculosis. In the group with complications 37.5% had vital capacities below 70% (Table XIII). This change is just about equal to the lowering of vital capacities among miners with coalescent pneumoconiosis (Table X).

Many roentgenograms read as classification A, B, and C (coalescent lesions), and some read as AX (suspected coalescent lesions), were also reported according to the American Public Health Association classification as "silicosis with infection," a total of 514 cases. All the films reported as silicosis with infection, together with the available history information, were further reviewed by a group of experts.

As a result of this review three sputum samples were collected from each of 89 examinees. Of these, only one sputum sample was returned from the laboratory as positive for tuberculosis. This confirms British reports that sputum examinations in cases with coalescent lesions are usually negative.

At the time of the writing of this paper 11 of the 18 persons reported with roentgenographic evidence of histoplasmosis or possible histoplasmosis had been skin-tested with coccidioidin, histoplasmin, blastomycin, and purified protein derivative by the Communicable Disease Control Division of the Pennsylvania Department of Health. There were four positive histoplasmin reactors in this group. Further studies in this area are contemplated.

Table XI—All Examined Miners Positive Medical History

Disease	Total Number of Cases
Tuberculosis	21
Pneumonia	791
Pleurisy	690
Heart trouble	260
Bronchial asthma	335
Hay fever or allergies	363

Discussion

Because of the voluntary participation in this study and the small percentage of the universe

Table XII—Miners Without Pneumoconiosis—Vital Capacity and Positive Medical History

Medical History	Percentage of Predicted 3-Second Vital Capacity					Total
	90% and Over	70-89%	50-69%	Under 50%	Unknown	
Tuberculosis	6	1	3	2	0	12
Pneumonia	208	160	63	48	24	503
Pleurisy	194	128	57	35	22	436
Heart trouble	66	46	32	17	4	165
Bronchial asthma	63	43	46	29	13	194
Hay fever and other allergies	114	62	35	16	18	245
Total	651	440	236	147	81	1,555
				24.6%		

Table XIII—Miners Without Pneumoconiosis—Vital Capacity and Complications

Complications	Percentage of Predicted 3-Second Vital Capacity				Total
	90% and Over	70-89%	50-69%	Under 50%	
Pleural abnormalities	236	227	116 (16.3%)	132 (18.6%)	711
Emphysema	7	15	11 (18.0%)	28 (45.9%)	61
Tuberculosis and suspected tuberculosis	5	4	1 (6.7%)	5 (33.3%)	15
Old tuberculosis	3	1	0	7 (63.6%)	11
Total	251	247	128 (16.0%)	172 (21.5%)	798

studied, it is difficult to draw conclusions concerning the absolute value of figures in the preceding tables. Participation of the union membership varied from 2% to 96% in the various locals. We will never know definitely whether the 25% of the universe who participated were a proper cross-section of the mining population of the area. The questions arise: Did most of those who felt that they had chest problems present themselves, or did a majority of the men who did have chest disease stay home? Even if all of the nonparticipants had negative roentgenographic findings, the percentage of those with positive findings would still be impressive.

Over-all, 28% of the population included in this study had roentgenographic evidence of pneumoconiosis in Category I or greater. This compares well with the prevalence of 24.8% found by McCallum and his associates[13] in Great Britain. Outhred and his co-workers[14] report a prevalence of 3% of pneumoconiosis among working miners in New South Wales. The prevalence of pneumoconiosis among working miners in our sample was 25%. The higher figure might possibly be due to the greater number of men in the upper age groups in our study.

The findings in the study of dust exposures in 14 mines of the central Pennsylvania coal district reported by Baier and Diakun elsewhere in this issue of the JOURNAL are incomplete and permit only tentative conclusions. It is fully realized that the dust concentrations and dust compositions found at the time of the survey are not necessarily representative of the exposures of the various individuals during their lifetimes. Of more than 300 dust samples collected in the working environment, 90% showed less than 2% of free silica. This finding gives rise to the hypothesis that either low concentrations of free silica or high concentrations of coal dust or a combination of the two play a significant role in the production of pneumoconiosis in these miners.

Tables I through IV illustrate the well-known fact that the prevalence of pneumoconiosis increases with age and number of years worked in the mines. This is due, of course, to the longer period of exposure in the older men. Only 9% of the working population with less than 20 years service had or were suspected of having pneumoconiosis on the basis of roentgen examination. In addition, 13 out of 1,168 working miners with less than 20 years' work in the mines were found to have coalescent pneumoconiosis. It would appear from these data that an exposure approaching 20 years is necessary to produce any significant degree of pneumoconiosis in our group. This period of time is in agreement with earlier observations made by Clarke and Moffet,[21] as well as Reed and his group,[16] that a majority of men with roentgenographic findings of pneumoconiosis give a history of having worked 15 or more years in the mines. This is in direct contrast to the relatively short period required to produce silicosis in workers exposed to higher concentrations of free silica, such as crystal grinders and granite workers.

Another interesting observation is that this study revealed 28 men with coalescent lesions superimposed on Category I pneumoconiosis. In contrast, Fletcher[17] states that his group had not seen progressive massive fibrosis on the background of less than Category II simple pneumoconiosis.

Tables V and VI and Figs. 12 through 15 confirm the observation made in several previous studies that there is poor correlation between roentgenographic findings and symptoms. Table VI shows an increase in the prevalence of cough with progressive severity of pneumoconiosis. This condition is further influenced by such factors as advancing age and number of years in the mines (Figs. 14 and 15). The influence of smoking was not evaluated for reasons indicated in the discussion of the survey. Newell and Browne[18] have drawn the conclusion that there is no evidence of association between symptoms and radiologic category for fixed age, and in this the authors of this report would concur.

Figs. 12 and 13 show that the degree of shortness of breath is proportional to age and number of years worked in the mines. An increase of dyspnea is expected with advancing age in the general population; therefore the amount of dyspnea for which mining is responsible cannot be determined without a control group. Levine and Hunter[19] show the presence of dyspnea in 61% of the men studied. In our study, the incidence was 49.9%.

The finding that respiratory disability increases with advancing age is illustrated in Tables VII through IX. This relationship has been observed by many investigators in the past.

Table X permits the same observation for this bituminous mining population as Motley[20] made in his study of diatomaceous earth workers, namely, that no apparent correlation was found between the pulmonary function measurements and roentgenographic findings.

Since completion of the analysis of the 5,072 miners' examinations of central Pennsylvania more than 10,000 miners in western Pennsylvania have been studied in the same manner.

The data for this group are now being tabulated. It is planned to continue these surveys to reach as large a percentage of Pennsylvania's bituminous mining population as possible in order to determine the exact incidence of lung pathology in this group. Arrangements are now being completed to study postmortem specimens of coal miners' lungs for free silica and other dust content to supplement the x-ray findings.

Extensive dust studies of bituminous mines are planned by the Pennsylvania Department of Mines and Mineral Industries. When these studies can be correlated with findings of the miners' survey, we hope to be able to make recommendations for the improvement of the working environment.

Conclusions

The only conclusion which can be drawn at this time is that there is a sizable amount of pathology resulting from exposure to dust in the central Pennsylvania bituminous mining population. Further studies are necessary to determine the exact prevalence of various pulmonary abnormalities.

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Pneumoconiosis Study in Central Pennsylvania Coal Mines

II. ENVIRONMENTAL PHASE

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FOR SEVERAL years the Pennsylvania Department of Health has been concerned with the dust exposure of persons employed in coal mines. A statistical summary[1] was prepared concerning compensation data, but no recent information was available concerning environmental data. Although limited information existed from studies of anthracite mines[2, 3], no reported studies had been made in the bituminous coal fields of Pennsylvania. Because of the absence of such information, the Pennsylvania Department of Health decided to undertake a study of the environment simultaneously with a medical study of coal miners.

Pennsylvania has both bituminous and anthracite fields and environmental studies were made in each. This report is concerned with 14 bituminous mines in Pennsylvania. The preceding article in this issue by Lieben, Pendergrass, and McBride containing the results of a medical study of 5,072 coal workers within this area is a companion report to this environmental study.

Geology

The workable coal beds (strata or seams[4, 5]) of the area under observation were in the Allegheny Group of Pennsylvania rocks[6, 7, 8] (see Fig. 1, Generalized Columnar Section Sketch). The coal seams lie horizontally for the most part, one over the other, with beds of shale, sandstone, limestone and underclays interlaced. Five of the minable beds and the vertical distances between beds, in descending order include:

1. Upper Freeport (Lemon or Four Foot)—45 to 70 feet
2. Lower Freeport (Moshannon)—20 to 65 feet
3. Upper Kittanning—31 to 45 feet

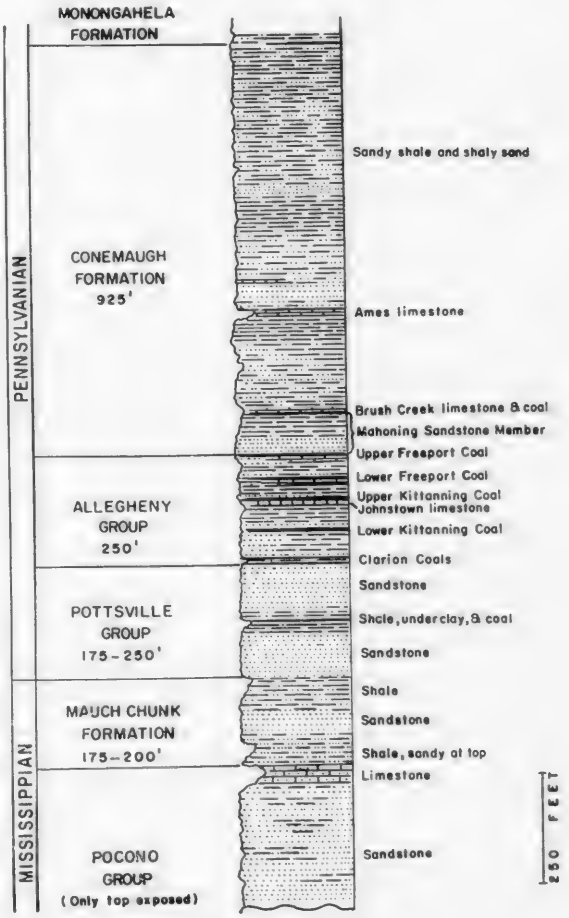

Fig. 1—Generalized columnar section.

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Fig. 2—Training course: studying roof structure.

Fig. 3—Training course: studying ventilation techniques.

4. Middle Kittanning—variable to 25 feet
5. Lower Kittanning (Miller B)—180 to 200 feet

The vertical distances between beds remain fairly constant in spite of changed elevations of any coal bed due to folding of the rock strata after coal deposition. This folding has produced a series of synclines (broad troughs) and anticlines (ridges) running northeast-southwest.

In general, the coals rise to an angle of 6 degrees.

The coals found in the area surveyed contain 15% to 25% volatiles on a dry, ash-free basis. The ash content is variable, but is usually between 6% and 12% in mined coal.

Study Preparation

The environmental survey team was composed of industrial hygienists, thoroughly trained in the surveying of industrial operations for hazards associated with them. Only a few had been associated with coal mines. To better acquaint these industrial hygienists with mining operations, a course (Fig. 2 and 3) was held at the U.S. Bureau of Mines Field Office at Pittsburgh. This course consisted of discussions concerning the main objectives of the environmental study (exposure of miners, exposure of miners from related operations, classification of occupations, and observation of dust control practices), related information on the mining operations (age of mine, mining methods, transportation, roof supports, and blasting practices), factors in dust control (use of water and ventilation), dust surveys, and laboratory procedures. This course was taught by mining engineers of the U.S. Department of Interior and by mine inspectors of the Pennsylvania Department of Mines and Mineral Industries. Literature obtained during this course* was circulated to staff members.

Following this training, meetings were held between representatives of the Pennsylvania Department of Health and the Pennsylvania Department of Mines and Mineral Industries. It was decided that a state mine inspector should accompany the industrial hygiene team on all surveys of environmental conditions. The industrial hygienist would select a mine, schedule the survey with the mine inspector, and conduct the survey in his presence.

This plan worked effectively. It placed a person on the survey team who was thoroughly familiar with all existing operations, health and safety conditions, and hazards of the mine. In utilizing this plan, the mine inspector was responsible for the safe conduct of the survey team while in the mine.

Equipment was provided adequately to protect the industrial hygienist. This included a miner's hat; a safety belt to hold the battery and accessory equipment; miner's lamp; and safety boots.

Mine transportation equipment presented many hazards. Extreme care had to be exercised at the face of the mine to keep clear of the mining machines as well as the miners.

The training continued after the completion of the first survey. This was conducted by a team of industrial hygienists who had become familiar with mining operations. As this team continued its survey, other industrial hygienists accompanied it. These, in turn, began additional surveys and also trained other industrial hygienists. In this manner, the staff of industrial hygienists received on-the-job experience and training.

It was deemed advisable to begin the study with those mines employing 50 or more miners.

* A list of this literature is contained in References 9 through 18.

The names and locations of such mines were obtained from the Pennsylvania Department of Mines and Mineral Industries, which collects, records, and compiles statistical data[19] for all coal mines in the Commonwealth.

In order to obtain sufficient information concerning the physical characteristics of the mine and its operations, the following form was developed:

Commonwealth of Pennsylvania
Department of Health
Division of Occupational Health

Name of company .....
Name of mine .....
Location of mine .....County.....
Arrangements for survey made with.....Title.....
State mine inspector accompanying.....
Survey dates .....
Company officials accompanying .....Title.....
.....Title.....
.....Title.....
Union mine: yes..... no..... Local #.....
Gassy mine: yes..... no..... How long so classed.....
Number of employees:
Grand total..... Total above ground..... Total below ground.....
Shift 1 above..... below.....
Shift 2 above..... below.....
Shift 3 above..... below.....
Average days worked per year.....
Number of hours worked per day: Shift 1 .....
Shift 2 .....
Shift 3 .....
Breakdown of time spent per day at different locations and/or operations
Average traveling time to work place.....
Average eating time.....
.....
Rockmen .....
Other .....
Age of mine..... Years operated by above company.....
Depth above / below sea level.....
Number of levels.....Vertical distance between levels.....
Level and number of men on each level.....
.....
Entry system:
Drift..... Slope..... Shaft..... Other.....
Method of coal transportation .....
Method of man transportation .....
Roof support:
Natural rock strata .....
Timber .....
Steel .....
Roof bolting .....
Other .....
Characteristics of coal bed: Av. thickness of vein.....
Degree of pitch .....
Coal production (average tons/day) .....
Mining methods used .....
.....
.....
% Continuous mechanical mining... water jets... local exhaust....
Dry / Supplementary / Wetted

Drilling coal:
% long hole .....
% manual auger .....
% rotary air machine with twist steel bits.....
% jackhammer .....
Other .....

Loading coal:
% manual mucking (shovel) .....
% gravity chute .....
% conveyor (tube) .....
% mechanical mucker .....
Other .....

Drilling rock:
What methods are used (wet drilling, dust collector, type drills, etc.) .....
.....

Loading rock:
.....
.....

Wet methods used:
Are the face, ribs, and bottom wetted before blasting.....
Are haulageways wetted down .....
Is the mine naturally wet..... elaborate.....
.....
Other wet methods used .....
.....
Is rock dusting used as a means of allaying dust.....
Ventilation:
Methods used to ventilate mine: .....
.....
How is the face ventilated .....
.....
Number of surface fans used..... capacity of each.....
.....
Number of inside booster fans and capacity of each.....
.....
Total amount of ventilation in use..... cfm
Respirators: % of miners who use them..... Approved:
Yes..... No.....
% of rockmen who use them..... Approved:
Yes..... No.....
Medical services:
.....
X-ray examinations—periodic..... pre-employment.....
Physical examinations—periodic..... pre-employment.....
Name of physician..... Aluminum therapy:
Yes..... No.....
Temperature range within mine: Winter ..... Summer: .....
Relative humidity range: Winter: ..... Summer: .....
Additional information .....
.....
.....
Comments .....
.....
.....
Sampling instruments used:
..... for .....
..... for .....
..... for .....
Survey by .....
.....

Sampling Methods

The principles of environmental sampling and control as applied in the field of industrial hygiene to industrial establishments can be readily transposed to mining operations. There are, however, several instances in which this transposition cannot be made directly. Because of the distances involved, the equipment used must be lightweight, compact, and sturdy. This equipment must also be operated without electricity owing to the lack of electrical outlets and the hazard associated with potential pockets of explosive gases. Since sampling operations are carried out at great distances from the source of equipment supply, great care must be exercised in preparing the descent into the mine. All equipment must be checked to determine its operating efficiency prior to entering the mine, because a faulty pump can result in a worthless trip to the distant sampling site.

Several problems were encountered in the selection of proper instrumentation. During earlier environmental studies of coal mines in the United States, the midget impinger was used.[20-25] This instrument was selected because its source of suction is manual rather than electrical and also because its use would permit one to compare the data obtained with those from earlier studies.

In Great Britain, the standard instrument is

Fig. 4—Collecting air sample for particle size analysis using membrane filter apparatus.

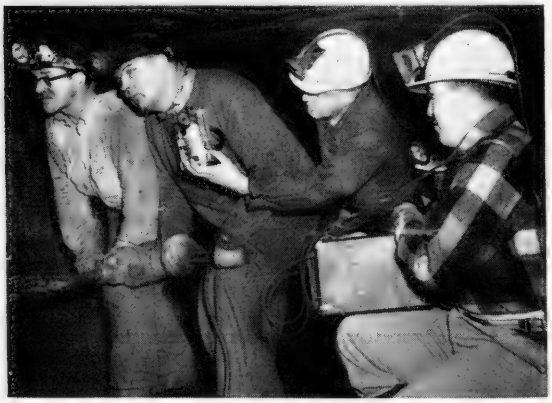

Fig. 5—Collecting air sample in breathing zone of rotary driller using Freon-propelled sampler.

the thermal precipitator.[26-29] A unit was obtained for use in this study so that available British data might be compared.

Several samplers not available to earlier investigators in coal mines were also utilized in this study. Membrane filters (Fig. 4) were used principally for particle size measurement of the airborne dust.* The use of these filters resulted in the collection of particles of a small size and without the possibility of fracturing during impingement. The filter also permitted deposition of particles in groups and flocs on the filter in the same state in which they existed in the mine atmosphere. While this could have been done with a thermal precipitator, the membrane filter was much more versatile. It was less fragile and cumbersome. Also, a Freon propellant assembly (Fig. 5) was used in place of the manually operated midget impinger pump as the study progressed. This unit could be held in one hand with the other available for support while sampling on inclined beds. Its small size permitted the industrial hygienist to carry additional sampling flasks.

In order to determine the reliability of the different sampling instruments, a study project[34] was designed. A midget impinger powered by a manual pump, a midget impinger powered by Freon, a thermal precipitator, and membrane filters were used to sample the mine atmosphere simultaneously.

The membrane filter was operated with a midget impinger pump or the Freon propellant. The standard size 47 millimeter filter holder was fitted with a 4.9 liter per minute limiting orifice, which when calibrated on a dry gas meter indicated a sampling rate of 0.05 cubic foot per minute using the manually operated pump.

The thermal precipitator operates on the principle that dust particles are rejected by a hot surface and are attracted by a cold surface. It samples airborne particles not in excess of 5μ in diameter with a high degree of accuracy. The sampling rate of the unit used in this study was approximately 7 cc per minute. Cover glasses were boiled in a mixture of equal parts of water and concentrated hydrochloric acid for cleaning prior to use.

It was found that dust counts with all four instruments were in fairly good agreement when a large number of samples were collected (Table I). From more than 50 sets of samples the mean dust concentrations, in millions of particles per cubic foot of air, were 6.91 for the impinger with a manual pump, 7.91 for the impinger propelled by Freon gas, 7.43 for the molecular filter, and 6.66 for the thermal precipitator.

Dust Counting and Sizing

In the bituminous coal mines surveyed, samples of airborne dust were collected by utilizing

Table I—Comparison of Dust Concentrations Determined by Various Sampling Instruments

Sampling Instrument	Mean Dust Concentration (millions of particles per cubic foot of air)
Impinger powered by manual pump assembly	6.91
Impinger powered by Freon gas assembly	7.91
Molecular filter	7.43
Thermal precipitator	6.66

* The technique for use of membrane filters is described in references 30 through 33.

Fig. 6—Slope entrance to a coal mine.

a midget impinger operated at approximately 0.1 cubic foot of air per minute. All impinger samples were counted within 24 hours by the standard light-field technique using a 10× objective, 10× eyepiece, and Sedwick-Rafter cells with a 20 to 30 minute settling time.

The dustiness is subject to considerable variability from moment to moment and from day to day even when the work appears to be done at a steady rate. The problem is complicated by the fact that the dust cloud to be sampled is not stable in space or time. A proper measure of dust exposure requires a picture of the variations in dustiness as well as a measure of the average concentration. To present this information is a difficult task. To formulate a complete picture of the levels of dustiness in bituminous coal mines, the median was chosen in this study as being more indicative than the mean for estimating miners' exposure. But in order that our data may be compared with those of earlier studies, both the median and the mean are presented.

If during the course of sampling the industrial hygienist collected airborne dust during periods of "dust peaks" and the results were averaged, the mean could be exceeded by most of the individual observations.

Since the median divides the set of observations in half, 50% of the observations smaller than the median and 50% larger, the middlemost observation is less affected by a single dust sample of very high or very low concentrations than is the mean.

Samples for subsequent size frequency determination were collected on membrane filters. The midget impinger pump, Freon-powered pumps, and aspirator bulbs were used to collect samples for sizing.

The respiratory system is selective in dealing with dust particles of different aerodynamic sizes. Particles larger than 5μ are efficiently trapped in the upper respiratory tract during inhalation and were not considered.*

Particle size was determined by optical microscope with use of techniques elsewhere described.[45-47] The determinations were made by measuring the diameter of a large number of individual dust particles with an eyepiece micrometer or a Mays-Porton graticule using a microscope with an oil immersion 95× objective and a 10× eyepiece. All particles from the smallest visible to 5μ in size were considered.

Environmental Study

The mines surveyed had been worked for 10 to 50 or more years. The longest main haulageway (main tunnel through which men and material enter and leave mine) was about 7 miles. Crosscut entries (tunnels driven at an angle to the haulageway) were connected to the main haulageway in a general fanlike pattern. Crosscuts were made at 50 to 90 feet intervals. In general, the coal bed was in a horizontal plane with occasional slight dips or rises. Access to the work area was gained through either a single slope (inclined passage) (Fig. 6), or drift (horizontal passage) via motor mine cars. A slope or drift is used for the purpose of ventilation, drainage, or for hoisting men and material.

Travel within the mine was by a small motor driven mine jeep (trackless vehicle) or a mantrip (track cars mainly used for transporting men into the mine). Other convenient methods for travel within the mine were riding in empty coal cars or on rubber conveyor belts. Conveyor belts from several work faces (points at which coal is being worked away) converge at a main belt which transfers the coal to rail cars. These belts extend as far as 2,000 feet into the entries. Because of the relatively great distance from the haulageway to the face, the belts are also used to transport miners. It took from 25 to 40 minutes to reach the working face from the entry.

The mining method depends mainly on the natural conditions of the mine and the size of the operation. These determine the mining method, the dimensions of the entries, the size of the rooms (area from which coal is mined) and pillars (solid blocks of coal for roof support), the manner in which the coal is cut and blasted, and whether manual or machine loading practices are observed.

In small mines, each crew (generally two

* References 35 through 44 contain American, British, and German reports on this subject.

men) at the coal face perform all the mining operations, i.e., drilling, cutting, blasting, timbering (installation of props used to support mine workings), and loading. Generally, drilling is done by an electric auger; however, some manual drilling is encountered. Cutting is done by the conventional machine undercutter (a machine used to cut below the face of the coal).

Table II—Comparison of U. S. Bureau of Mines Studies and Pennsylvania Department of Health Study for Dust Generated During Wet Undercutting Operations

Study	Dust Concentration (millions of particles per cubic foot of air)		
	Mean	Min.	Max.
U. S. Bureau of Mines	21.8	3.6	42.9
Pennsylvania Department of Health	22.5	2.4	73.1

Undercutting coal is an extremely dusty operation and the dust clouds produced are frequently very dense. This is especially true in dry coal beds. In 1927 and 1937 the U.S. Bureau of Mines conducted studies[16, 48] in the bituminous coal fields in Pennsylvania, West Virginia, and Alabama and found that the largest total amount of dust was generated by dry undercutting. The quantity of dust raised into the air during this process ranged from 65.5 to 163.9 million particles per cubic foot of air, averaging 120.9 million particles per cubic foot of air. When water was used on the mining machines, the dust ranged from 3.6 to 42.9 million particles per cubic foot of air, averaging 21.8 million particles per cubic foot of air.

Water was used to reduce the rate of dust generation at all undercutting operations evaluated during this study. Concentrations ranged from 2.4 to 73.1 million particles per cubic foot of air. To compare our findings with those of the U.S. Bureau of Mines, our mean was found to be 22.5 million particles per cubic foot of air (see Table XIII). Since these findings agree (Table II), it may be stated that the degree of dustiness seems to be the same for undercutting.

The primary method of moving coal from the face to the mine cars in the haulageway is by shaker chutes (vibrating conveyors) which are loaded manually.

In the larger mines surveyed, all operations were mechanized and each miner performed one specific duty. After machine undercutting and blasting, mechanical loaders were utilized to load shuttle cars which moved the coal to continuous conveyor belt headings (continuous passageways).

Shot Firing

Blasting operations[1, 5] or shot-firing as it is sometimes referred to, is ordinarily conducted in sub-surface coal mining. The methods employed in breaking down the face, which often includes rock as well as coal, are the conventional dynamite, airdox (compressed air applied to a cylinder until the cylinder ruptures), and carbon dioxide (expansion of liquid gas until the containment ruptures) methods. Holes are either drilled with an electrically powered auger type bit or the drilling is done manually. The extent of mechanization in a particular mine determines the method employed, but generally power drills are utilized. The placement of shot holes, types of blasting materials used, and method of blasting depend on natural conditions existing within the mine.

In development work (the opening of shafts, passageways, etc., to reach coal beds), the coal is blasted separately from the rock. Occasionally a rock stratum is incorporated in a coal bed and is broken down in conjunction with the coal. The number of shots average about six per working face.

Shot firing may be done by an individual miner whose sole duties are preparing and detonating charges, or it may be done by an individual or a member of a team who performs other mining operations as well.

The dust exposures of shot firers resulting from dust generated by the blasting operations alone is dependent on the nature of their duties. This is evident from the range of dust concentrations found after blasting—1.5 to 734 million particles per cubic foot of air (Table XIII). The high concentrations were obtained when the men returned to inspect the face without sufficient time to permit settling and/or removal of the dust by ventilation. Some of the airborne dust resulted from agitation of settled dust being regenerated into the mine atmosphere during the blast.

A mean concentration of 91.4 and a median of 37.9 million particles per cubic foot of air were recorded. In the absence of good ventilation, shot firing produces a large quantity of dust, but only for a short period of time. With proper ventilation, the dust produced is diluted and removed. In a few instances respiratory protective devices were employed by the personnel associated with the operation.

Coal Loading

After the coal is broken, it is loaded manually or mechanically into mine cars and hauled to

the outside by motor or other haulage. Some of the mines surveyed use mechanized mining methods, where the coal is cut, drilled, loaded, and hauled by mechanical means. A recent method, called "continuous mining," combines all of the above operations in one machine. The "continuous mining" method has many manufacturers' trade names, but it uses the principle of digging and loading coal by circular arm action (continuous revolving chains or wheels mounted with sharp bits or rotary drills or augers). The loading rates vary with the hardness of the coal, impurities in the seam, and the conditions of the roof and floor. The principle of "continuous mining" is cutting or breaking the coal while advancing, and at the same time loading it into shuttle cars or conveyor lines, or depositing it on the mine floor to be loaded by a machine. During continuous mining operations roof support is achieved by roof bolting (Fig. 7). The automatic loader is a trancing type of machine with a large gathering head composed of controlled moving arms used to feed coal or rock to a straight-through conveyor mounted on top of the machine. The above procedure was found in some of the mines surveyed.

In pillaring operations, all of the coal is recovered. This is a retreat system of coal mining. The pillars or stumps are areas of coal left for support of the roof. When advance or developmental mining is halted, the pillars are then mined out or pulled. As the coal is mined, the large open areas collapse, easing the excessive pressure of the overburden on other areas of the mine.

Loading operations are relatively dusty. The majority of the samples were collected without any reference to the moisture content of the material loaded. In a few instances a comparison was made between loading dry coal and wet coal: a reduction in the levels of dustiness was found. Table III contrasts the concentrations of 10 samples found during uncontrolled working conditions and during the use of wetting and dust collectors.

As can be noted from Table III, loading coal by conveyor, using wet methods, reduced the dust load by a factor of 4. This reduction is also evident when roof drilling operations are considered. A 20-fold decrease is noted by the use of a simple collector affixed to the drill.

Although all of the continuous type mining machines were equipped with sprays, and most mines carried ventilation to the face, the dust concentration found averaged 41.9 million particles per cubic foot of air. The median was 31.6 million particles per cubic foot of air (Table XIII).

Fig. 7—Collecting air sample of a miner's dust exposure during roof bolting.

Sampling during shuttle car transfer of material averaged 33.5 million particles per cubic foot of air, with a median of 22.9. During loading, an average value of 40.4 and a median of 18.4 million particles per cubic foot of air was obtained (see Table XIII).

Control measures in use to allay dust are wetting, ventilation, and removal of fines by manually shoveling machine cuttings onto a car from the work area.

All the mines were naturally wet. In slope mines it was necessary to pump water from the haulageways and entries. However, occasional dry pockets occurred in the coal bed with resultant high dust concentrations during mining operations. To control dust exposure in these areas, supplementary wetting methods were occasionally employed. The magnitude of the dust concentrations did not appear to be dependent so much upon the mining method used as on the natural wetness of the seam and frequency of dry pockets.

Tipping

The tipping of mine railway cars may occur either above ground or below the surface, depending on the degree of mechanization neces-

Table III—Results of Comparison Between Operations in Bituminous Coal Mines Under Uncontrolled and Controlled Conditions

Operation	Dust Concentration (millions of particles per cubic foot of air)		Control Measure
	Uncontrolled	Controlled	
Loading Coal (belt conveyor)	219.0	53.3	Wetting loaded material
Roof drilling in rock	235.0	21.6	Dust collector
Roof bolt drilling	455.0	20.6	Dust collector

sary for economical operation of the mine. The contents dumped from the cars are either directed down gravity chutes or moved by conveyors. Mine waste is transported to a dumping area, and coal is transported to a cleaning house.

Complete wetting of the materials in cars before dumping aided greatly in reducing dust generation in this operation. Dry tipple dumping operations near the mine air supply may increase dust concentrations throughout the mine by several millions of particles per cubic foot of air.

All of the air samples collected at one tipple were from operations handling dry material. The mean and median dust quantities found were 33.9 and 34.8 million particles per cubic foot of air respectively. These are comparable to the dustiest operations within the mine.

Ventilation

The basic type of mine ventilation system consisted of one or more large volume fans, located at or near the surface of an airway exit tunnel or shaft. The fans were so located as to maintain a negative pressure throughout the mine. This condition caused a constant flow of air from above ground through entry tunnels and shafts and airway tunnels and shafts through working areas and then to the fans. All headings and tunnels not in use were sealed off by partitions to guide the airflow through work and use areas of the mine. Bratticies (wood forms covered with burlap to channel the airflow) were erected where necessary.

These were installed along conveyors, shaker and belt, car loading stations and at the face to remove dust laden air from points of high dust generation. Supplementary air moving equipment, including compressed air blowers and ejectors, was sometimes used in the mine to generate higher airflows through bratticed areas to the working face.

For practical ventilation evaluation, a mine may be considered as a complex ductwork with all mining operations performed within the ducts. In evaluating the ventilation rates a stop watch, a rotating vane anemometer or a smoke tube, and a reel tape measure were used. The method employed was as follows:

1. Selection of points of measurement
   (a) In the main haulageway at sufficient distance from the entrance to avoid the turbulence created by entry of air through the throat of the system
   (b) In crosscuts at sufficient distance from entries or rooms
   (c) At bratticies
   (d) At the working face
2. Measurement of sites at point of velocity measurement.

Fig. 8—Conducting air velocity traverse of haulageway using a vane anemometer.

Fig. 9—Determining air velocity by means of a smoke tube.

The rotating vane anemometer was used to take measurements of a one-minute traverse across the entire location (Fig. 8). If the area was greater than 15 feet in width, a one-minute traverse was made for each half area, and the velocity measurements were averagd. In areas of low air velocity, smoke tubes were used (Fig. 9). Two men were positioned at fixed distances. The industrial hygienist upstream released a smoke cloud from a smoke tube. The second industrial hygienist timed the movement of the cloud downstream until the middle of the cloud reached his position. The air velocity was determined by dividing the fixed distance by the time.

In all mines surveyed the air supply conformed to present mine laws.[49] These laws require a minimum of 150 cubic feet of air per minute for each man underground in nongaseous mines and 200 cubic feet of air per minute in gaseous mines. The rated capacity of the surface fans in use at the mines surveyed ranged from 23,000 to 500,000 cubic feet of air

per minute. Measurement of ventilation rates at the face ranged from 900 to 20,000 cubic feet of air per minute. It was not a simple task to determine the actual volume of air supplied to each workplace. This can be appreciated when one considers the irregularities of the work areas and the difficulty in distinguishing between useful and turbulent currents.

Table IV—Dust Exposure of a Miner Engaged in Machine Undercutting

Activity and Remarks	Dust Concentrations (millions of particles per cubic foot of air)	Average Concentration (millions of particles per cubic foot of air)	Time Spent at Each Activity (minutes/day)	Particles Minutes (millions per cubic foot of air)
Motor trip into and out of mine	6.6 5.7	6.15	45	277
Conveyor belt trip to and out of crosscut	14.1 19.7	16.9	45	761
Waiting for supplies	0.5	0.5	45	23
Moving machine to new work face	1.6 2.3	1.95	252	491
Undercutting and rotary drilling coal face	19.9 25.5 54.0	33.1	73	2416

$$\text{Weighted dust exposure for work day} = \frac{3{,}968 \text{ million particle minutes}}{460 \text{ minutes}} =$$

8.63 million particles per cubic foot of air

Table V—Dust Exposure of a Motorman

Activity and Remarks	Dust Concentrations (millions of particles per cubic foot of air)	Average Concentration (millions of particles per cubic foot of air)	Time Spent at Each Activity (minutes)	Particle Minutes (millions per cubic foot of air)
Trip into mine work areas, aboard 1st car behind motor, cars empty	3.1 0.7	1.9	120	228.0
Time spent by motorman inside mine when trip not moving				
General air	0.4	1.47	120	176.4
Haulageway, 50 ft. from face	1.2			
Car loading by conveyor belt	2.8			
Trip out of mine, trip loaded with coal aboard last car	2.3	2.3	120	276.0
Time spent by motorman outside mine between trips, includes eating time				
Crosscut entry	0.5	0.5	120	60.0
Main entry	0.5			

$$\text{Weighted dust exposure during work day} = \frac{740.4 \text{ million particle minutes}}{480 \text{ minutes}} =$$

1.54 million particles per cubic foot of air

Weighted Exposure

After reviewing and examining preliminary coal mine reports, it was decided to conduct surveys in order to obtain the miner's weighted average exposure[50, 51] to airborne dust. This was done to evaluate dust concentrations determined during site sampling. During the preliminary coal mine survey several airborne dust samples, bulk samples, and membrane filter samples were collected at representative working sites within the mine. A weighted exposure survey was made to determine the average amount of dust to which an employee was exposed during his working day. In order to accomplish this, it is necessary to understand the degree of dust exposure and the time during which an employee is exposed. By mathematical computation a single quantity may then be assigned, designating his average daily exposure. This procedure is known as a "weighted exposure" calculation.

A team of miners, usually two or three men, were chosen at random on the morning of the survey. They were accompanied for the entire shift. Airborne dust samples were collected at various intervals during the miners' working shift, every time their dust exposures changed.

In many coal mines a mining team works at one location for an entire day during which time it performs all operations related to mining. The time spent in each location and/or operation was recorded with other pertinent data such as the mine's method of mitigating the dust, the length of time and at what operations the miners wore respirators, the temperature and relative humidity of the mine, and the ventilation rates in the haulageway, crosscuts, and locations where the miners worked.

After the dust samples were counted, the miner's weighted average daily exposure was calculated by the following formula:

$$\text{Weighted average daily exposure} = \frac{E_1T_1 + E_2T_2 \ldots E_nT_n}{T_1 + T_2 \ldots T_n}$$

where E represents the atmospheric concentration to coal mine dust at n different jobs, and T represents the time in minutes engaged at each task.

Examples of the calculated dust exposures

for three occupations are presented in Tables IV, V, and VI.

The quantities 8.63, 1.54, and 5.82 million particles per cubic foot of air in these particular examples represent a summation of the dust dosage to which the miner would be continuously exposed during a working day.

It is indicated from Table VI that shoveling on to the conveyor belt contributed 55 % of the daily exposure to dust for 73 % of the time, and shooting, which required less than 1 % of the time, produced 16 % of the total dust exposure.

Table VI—Dust Exposure of a Roomworker

Activity and Remarks	Dust Concentrations (millions of particles per cubic foot of air)	Average Concentration (millions of particles per cubic foot of air)	Time Spent at Each Activity (minutes)	Particle Minutes (millions per cubic foot of air)
Walking into mine	0.4 1.3	0.85	15	12.8
Manual shoveling onto conveyor belt	4.4	4.4	350	1540.0
Drilling holes preparatory to shooting	10.2	10.2	30	306.0
Undercutting face prior to shooting	9.7	9.7	30	291.0
Shooting 7 dynamite charges	94.0	94.0	5	470.0
General air in room during break in operations, includes 30 minutes for eating	4.7	4.7	35	164.5
Walking out of mine	0.4 1.3	0.85	15	12.8

$$\text{Weighted dust exposure during work day} = \frac{2{,}797.1 \text{ million particle minutes}}{480 \text{ minutes}} =$$

5.82 million particles per cubic foot of air

The occupational analysis yields an estimation of the exposure and reveals the most important activity with respect to dust hazard. A total of thirteen weighted dust exposures were performed for eleven mine occupations; however, too few dust exposure studies were done to reach any statistically valid conclusions. Table VII contains a list of the weighted exposures found for several different occupations.

To demonstrate the comparison, weighted exposure data were calculated for motormen at two different mines, unrelated geographically. These were found to be 1.54 and 1.3 million particles of dust per cubic foot of air per day. For shuttle operators in two different mines, weighted exposure calculations of 6.2 and 6.1 million particles per cubic foot of air were calculated. In mining coal, the miners frequently change locations from one level to another. However, by comparing preliminary survey results with weighted exposure results, it appears that some correlation can be made between the amount of airborne dust at specific operations and the amount of dust to which a miner is exposed.

Table VII—Table of Weighted Exposures of Miners Engaged at Various Occupations Within the Coal Mine

Occupation	Weighted Dust Exposure (millions of particles per cubic foot of air)
Motorman	1.54
	1.3
Rockman	2.49
Roomworker	5.82
Conveyor car loader	1.71
Continuous miner operator	23.3
Shuttle operator	6.2
	6.1
Belt conveyor operator	129.6
Undercutter	8.6
Shot firer	137.0
Automatic loader	20.7
Roof drillers	61.7

Free Silica Analysis

Since the geology of the rock formations encountered in coal mines gives rise to dusts of varying proportions of materials in the various dust generating and dispersing operations in the mine, a sample of the coarse materials could not be assumed to represent that which could become airborne. To eliminate a potential source of error and obtain a representative sample, a clean white envelope was held out of the direct line of fall of materials arising from primary dust generating operations, such as drilling and undercutting, until a sufficient quantity of the material had been collected. During shoveling and loading coal cars, a sample of the coarse material was placed in the envelope. In the laboratory the larger particles were removed by 200 mesh screening.

The samples collected during this study were coal dust, rock drilling dust, and traction material. Most of the samples were coal dust.

The procedure followed for the analysis of free silica was that described by Talvitie.[52] The laboratory used supplementary petrographic techniques described by Landry[53] as a check. This technique also was used to verify the presence of free silica, when a sample indicated a low free-silica concentration.

The analysis of bulk dust samples as collected

from various mining operations was probably not a true indication of the free silica which is airborne and respirable. To collect enough sample, a high-volume sampler is required. However, explosive gases which make use of this instrument* impractical may be present in mines. An attempt was made to measure airborne quantities of free silica by collection on membrane filters. Laboratory efforts involving the determination of free silica mixed with coal dust deposited on a membrane filter showed that such a technique is possible with the use of petrographic methods. A serious disadvantage is the loss of sample by abrasion or vibration, even when edge-holding filter containers are used, during shipment of the sample to the laboratory. Because of this disadvantage further attempts to develop this technique were abandoned.

The free silica content of the dust found is presented in Table VIII. The traction material, used to prevent slippage of trip wheels on inclines, was primarily silica sand, containing approximately 87% free silica. When this material was dispersed into the haulageway, it contributed only an additional 1% free silica to the free silica already present. As may be expected, rock drilling was the greatest source of free silica exposure. In general, bituminous coal miners are exposed to dust that contains approximately 2% free silica.

Table VIII—Average Free Silica Content of Dust Found in 14 Central Pennsylvania Bituminous Coal Mines

Operation or Location	Free Silica (average percentage)
Haulageway	
General air	3.0
Traction material	87.0
Continuous miner	2.0
Undercutting	2.0
Drilling	
Coal	2.0
Rock	48.0
Afterblast	2.0
Loading coal	1.0

Mine Temperature

All relative humidity determinations were accomplished by utilizing a bulb aspirator psychrometer. Table IX lists the humidity found in some of the mines surveyed.

Very little difference was noted between summer and winter temperatures within the mine.

Table IX—Temperature and Humidity Observations Found in Some Bituminous Coal Mines

	Dry bulb Temperature (F°)	Relative humidity (per cent)
Minimum	53	65
Maximum	64	95
Mean	56	86

Dust Exposure Summation

During this study more than 7,000 particles were measured; these were reported for various operations and locations in the mine. The median particle size and the standard deviation were determined.[45] When all the median particle size measurements were averaged, a value of 0.89μ was found. The average frequency distribution of coal mine dust in various ranges is presented in Table X.

From these limited data the size of the mine dust measured fell into very narrow limits, an average of 94% being between 0.3 and 1.5μ. Approximately 68.5% of the particles were below 1μ, and 17% in the range 0.3 to 0.5μ.

Table X—Average Size Frequency Distribution of Dust from Fourteen Bituminous Coal Mines

Size Range (microns)	Average Frequency (per cent)
0.30 – 0.49	17.1
0.50 – 0.99	51.4
1.00 – 1.49	25.7
1.50 – 1.99	2.9
2.00 – 2.49	2.9

By calculation the smallest particle visible to the average observer using an oil immersion objective is approximately 0.3μ. Particles smaller than 0.3μ could not be sized by use of this technique.

Sayers, Bloomfield, Dalla Valle, Dressen, and Britten[2, 3] in their study in the anthracite coal fields of Pennsylvania reported a median particle size of 0.9μ. An average of 8% of the particles were below 0.5μ, 80% of the dust was found to be between 0.5 and 1.5μ. The difference between the percentages of particles in the range below 0.5μ in the present study and in the past study in the hard coal fields is due possibly to the method of sampling. The Owens jet dust counter was used in the anthracite study; membrane filters were used in this study.

In spite of the differences in the percentage of particles less than 0.5μ found in this study and that found in the previous study in the anthracite coal field,[3] it is believed that the dust generated in the bituminous coal mines today is essentially of the same size distribution as found in the anthracite industry.

* The U.S. Bureau of Mines has developed a high volume sampler with an explosion-proof housing, but this instrument was not commercially available at the time of this study.

Mine dust size may differ markedly from one situation to another as illustrated in Table XI.

An attempt was made to correlate the particle size distribution with ventilation rates found in the various mines surveyed. The premise is that the greater the total amount of ventilation, the greater the percentage of larger particles found in the mine; the smaller particles being preferentially removed by air currents. However, the attempt was unsuccessful, either because of inadequate ventilation measurements or, as Patterson[54] suggests, because small particles are held more firmly by cohesive forces to the surface on which they have settled.

A summary of dust concentrations found in 14 bituminous coal mines surveyed is presented in Table XII. It is evident from this Table that the dust levels vary considerably from mine to mine. The range of dust for all the mines surveyed was from 0.1 to 734 million particles per cubic foot of air.

For determination of the amount of dust to which the mine workers were exposed, 359 atmospheric dust samples were collected. The frequency distribution of the dust concentrations shown by the samples obtained is illustrated in Fig. 10. The median dust concentration found was 10.1 million particles per cubic foot of air, with a standard deviation of 5.0.

Throughout this paper, dust concentrations were reported in those sections under discussion. In order to present a tabulation of dust concentrations found in bituminous mining operations, Table XIII is presented.

Table XI—Variability of Size Distribution of Dust Particles by Operation In Bituminous Coal Mines

Operation	Average Median Particle Diameter (microns)	Average Standard Geometric Deviation
Planer	1.009	2.071
Air chisel	0.990	3.020
Drilling coal, dry	0.557	3.018
Drilling (rock)	1.240	1.980
Afterblast	0.592	2.612
Undercutting	0.626	2.193
Shaker loading	0.542	2.933
Continuous miner	0.756	2.039
Shuttle car	0.822	1.981
Shoveling coal	0.756	1.685
Haulage	0.715	2.097

Table XII—Dust Concentrations Obtained in Bituminous Coal Mines in Central Pennsylvania (359 samples)

Mine	Dust Concentration (millions of particles per cubic foot of air)			
	Mean	Median	Minimum	Maximum
1	51.7	39.0	0.1	335.0
2	51.8	47.0	18.8	81.4
3	24.5	24.2	11.2	40.4
4	81.5	51.7	0.5	734.0
5	14.1	9.1	0.6	56.0
6	6.1	4.8	0.8	23.8
7	8.7	5.8	0.2	38.4
8	30.7	14.7	0.5	208.0
9	77.2	53.3	10.0	205.0
10	37.1	30.5	3.2	75.0
11	39.9	22.0	2.4	185.4
12	29.7	5.8	0.6	250.0
13	3.5	2.4	0.2	9.3
14	20.4	17.8	0.8	70.0

From this Table, according to median concentrations, it appears that undercutting, afterblast, shuttle loading, and continuous mining were the dustiest operations in subsurface mining in that order. However, if the percentage of time expended per day at each operation is taken into consideration, continuous mining, mechanical loading, undercutting, rotary drilling, manual loading, shuttle loading, blasting, belt conveying, and manual drilling are the dust producers in descending order. This is presented in Table XIV. These data represent only operational exposure per se, and no attempt is made to extend these calculations to a weighted exposure of the miner. In order to determine a weighted exposure, each miner

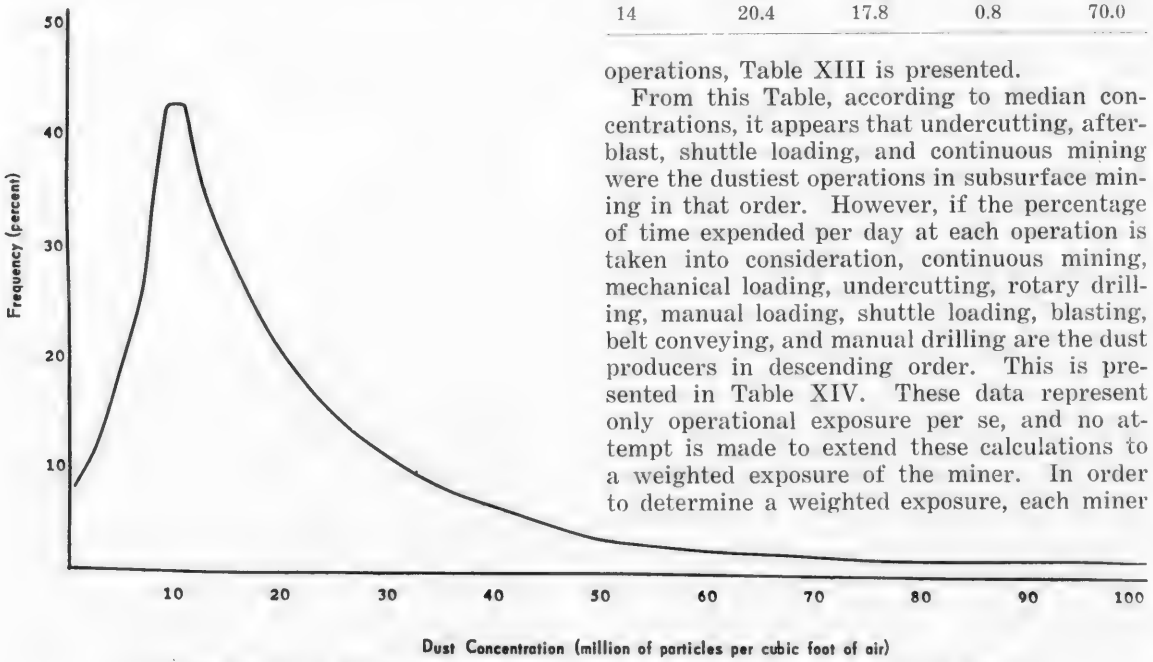

Fig. 10—Frequency distribution of dust concentration found in 14 bituminous coal mines.

must be followed. In these mines one miner was engaged in several operations. In the Table the dust concentration in the haulageway was not considered. The time reported is not absolute; it is but an approximation.

An interesting observation from this study is the comparison of data between fully mechanized and nonmechanized bituminous coal mines. Of the 14 mines surveyed, 6 were fully mechanized, and 8 were nonmechanized. Mines which operated by means of continuous mining equipment were considered to be fully mechanized. Those which conducted the mining of coals principally by manual techniques, even though equipment such as an undercutter was employed, were considered to be nonmechanized. There was an insignificant difference of median dust concentrations (Table XV) between the types of mining.

Table XIII—Summary of the Dust Exposure in 14 Bituminous Coal Mines

Operation or Location	Number of Samples*	Dust Concentration (millions of particles per cubic foot of air) Mean	Median	Minimum	Maximum
Undercutting	33	22.5	40.4	2.4	73.1
Afterblast	21	91.4	37.9	1.5	734.0
Mechanical loading	18	28.2	36.7	5.6	91.9
Continuous miner	35	41.9	31.6	1.7	192.0
Drilling (rotary)	24	82.7	26.1	0.5	297.0
Shuttle unloading	14	33.5	22.9	0.6	107.2
Shuttle Loading	22	40.4	18.4	2.5	205.6
Belt conveyor	49	37.4	5.3	0.2	321.0
Loading (manual)	29	40.5	22.7	0.9	185.4
Drilling (manual)	10	9.1	8.9	1.4	20.6
Tipple operation	8	33.9	34.9	11.2	54.4
Haulageway	73	4.4	3.0	0.1	38.0
Mine entrance	11	2.0	1.2	0.2	8.3
Mine exhaust fan	8	9.9	4.3	1.2	25.6

* Does not include samples collected which were not classified, i.e., break in air hose, etc.

To expand this comparison, location and certain operations within fully mechanized and nonmechanized mines were tabulated with dust concentrations (Table XVI). Although haulageway concentrations are in agreement, the analysis of operations seems to indicate that for the 14 mines surveyed nonmechanized mines produce a greater dust concentration.

Table XIV—Calculated Operational Dust Exposure of Bituminous Coal Workers by Time Distribution of Activity

Operation	Percent of Total Time	Median Dust Concentration (millions of particles per cu. ft. of air)	Relative Weighted Dust Exposure (millions of particles per cu. ft. of air)
Continuous miner	62	31.6	19.6
Mechanical loading	40	36.7	14.7
Undercutting	32	40.4	12.9
Rotary drilling	45	26.1	11.8
Manual loading	35	22.7	7.9
Shuttle loading	38	18.4	6.9
Blasting	10	37.9	3.8
Belt conveyor	46	5.3	2.4
Manual drilling	12	8.9	1.1

Comment

During this study we have surveyed bituminous coal mines from pick and shovel operations to modern mechanized techniques. Some of the pick and shovel type mines produced greater dust exposure to the miners than did some of the mechanized mines. On the basis of these observations it does not appear that the overall dustiness of a mine has changed significantly during the past several decades.

There are too many variables to correlate the type of mining method with the dust exposure of the miners. While total ventilation rates were in accord with mining codes, local ventilation rates in certain areas often were found to approach "still air" conditions. The dust concentrations as determined by standard dust collection and analytical techniques are not representative of actual exposure in many instances. The efficiency of each respirator used, its fit and filter medium cannot be determined.

Even in a wet mine with a wet face, the possibility of hitting a dry pocket with a drill or undercutter exists. The converse is also true. Several operations, such as the use of compressed air to blow out drill holes, tend to contaminate areas of low dust production. The movement of equipment, not necessarily a production procedure, and the movement of the miners themselves tend to redisperse settled dust and alter ventilation rates. Operation techniques for similar operations vary greatly in the quantity of dust produced or dispersed.

In this 14 mine study the principal sampling method for dust determination was the impinger. We began on the premise, accepted by many in this field, that the choice of instrumentation should be guided by the equipment used in previous studies. Originally, this study was conceived as an extension of limited earlier work. By using the impinger we had hoped to add to earlier data as an estimate of miners' exposure. Many persons, currently employed, had been exposed to dust in the mines for decades. An analytical review of the findings

Table XV—Comparison of Fully Mechanized and Nonmechanized Bituminous Coal Mines

Type of Mine	Number of Mines	Dust Concentrations (millions of aprticles per cubic foot of air) Mean	Median	Minimum	Maximum
Fully mechanized	6	29.3	9.8	0.2	205.6
Nonmechanized	8	41.3	11.3	0.1	734.0

in this study indicates that dust levels have not significantly changed in the mines with mechanization. Also, since we found a correlation[34] between impingers, membrane filters, and the thermal precipitator, we can make comparisons with British data and continue studies with a wider choice of instrumentation.

The nature of certain operations precluded an accurate estimate of miners' exposures. The survey team could not gain access to all locations. For instance, during pillar robbing only a few experienced miners are at work, and only those so engaged are permitted in the area. Additional persons would increase the degree of hazard. During this time the height of free fall contributes abnormal quantities of dust into the miners' breathing zones. It has not been possible to evaluate their individual exposure. At best a general air sample taken at a great distance from the source of dust generation must be used.

Summary

The results of an environmental study of 14 bituminous coal mines in Pennsylvania are presented. Dust levels for various operations and locations within the mines are tabulated. Size frequency of the dust agrees with previous studies and indicates that the dust is within respirable limits. Weighted dust exposure, in addition to random sampling as a means of classifying hazardous operations within an occupation are reported. The extent to which control measures are employed and their effectiveness in allaying dustiness are included.

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Table XVI—Comparison of Fully Mechanized and Nonmechanized Bituminous Coal Mines by Operation or Location

Operation or Location	Fully Mechanized Mines Dust Concentration (million particles per cubic foot of air) Mean	Median	Minimum	Maximum	Nonmechanized Mines Dust Concentration Mean	Median	Minimum	Maximum
Haulageway	4.4	2.4	0.2	23.9	6.3	2.8	0.1	38.0
Belt conveyor	13.1	5.3	0.3	73.3	50.1	19.2	0.2	321.0
Undercutting	10.0	8.3	3.0	27.0	30.7	26.2	2.4	73.1
Continuous miner	41.9	31.6	1.7	192.0				
Mechanical loader	28.2	36.7	5.6	91.9				
Manual drilling					9.1	8.9	1.4	20.6
Rotary drilling					82.7	26.1	0.5	297.0
Manually loading					40.5	22.7	0.9	185.4
Loading shuttle car	40.4	18.4	2.5	205.6				
Unloading shuttle car	33.5	22.9	0.6	107.2				

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The Disabled in Industry

—EXPERIENCE AT ABILITIES, INC.

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UP TO THE turn of the 20th century, the handicapped person was largely left to his own devices. Usually, he became dependent upon society for his care. It is only within the last 50 years that measures have been initiated to limit and correct the disabilities that were causing invalidism. The recent strides that have been made owe much to the understanding and direction of such centers as the Institute of Physical Medicine and Rehabilitation and the Kessler Institute and federal agencies such as the Office of Vocational Rehabilitation, the President's Committee on Employment of the Handicapped, and the Department of Labor.

The efforts of physical medicine now are directed toward retraining the handicapped person to live and work at his maximum functional capacity—to walk, to care for his daily needs, to apply and use the necessary prosthetic devices, to use normal methods of transportation, and to communicate either orally or by writing. All of these are necessary if the handicapped person once more is to attain his status in society as an independent and productive being.

Progress has, by and large, been successful. Men and women who were previously condemned to a wheel chair or bedridden existence are now being rehabilitated so that they can, if given the opportunity, attain independence. The rehabilitation is a continuum which consists of medical evaluation, treatment, training or retraining, experience in sheltered workshops, work in special shops, or work in competitive industry. But a good many of the disabled have fallen short of their goal of complete independence because of their inability to find work. Physical criteria for ability to work are still very stringent and in the main follow the "anatomic concept" of medicine, namely, that man in order to be competent to work must be free of anatomical defects. Thus, many of the handicapped who are functionally competent to work are denied employment.

Impetus for the employment of the physically handicapped was given by the Vocational Rehabilitation Acts of 1954. In these Acts, a physically handicapped person was defined as an individual with a physical disability severe enough to constitute a serious problem in securing employment. The disability must be of a somewhat static nature, and there must be reasonable expectation that vocational rehabilitation would render the person fit for remunerative employment. Prior to 1954, approximately 60,000 persons yearly were sufficiently rehabilitated to be employable. The Acts of 1954, through increased state and federal funds, have brought this goal within reach of an estimated total of 28,000,000 persons in need of rehabilitation. But, while the Acts have made great strides to increase the manpower available to industry, industry has not been entirely successful in absorbing these people. Yet, progress has been made toward the ultimate goal—the employment of all the handicapped who are, within the limits of their regained abilities, fit to work.

Abilities, Inc.

A company which employs only the seriously disabled was founded in 1952 by Henry Viscardi to prove that the disabled could work in a highly skilled enterprise as productively as the nondisabled worker. At the time Abilities, Inc., was founded, it consisted of five men who among them had one leg and five arms. Since then, it has grown to include more than 350 employees. Its physical plant has expanded from a 2,000-sq-ft garage to a 40,000-sq-ft air-conditioned plant. Its assets have grown ac-

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cordingly, so that its sale of products is now several millions of dollars annually.

The employees work at many different kinds of jobs in five manufacturing departments:

Harness and cable department—produces harness and cable assemblies used in aircraft firing systems, radar units, guided missile control panels.

Electronic department—assembles amplifiers, printed circuits, resistor boards, etc.

Packaging department—performs preservation packaging of such items as aircraft parts, parachute packs, electronic components, and spare part kits.

Motor and coil-winding department—manufactures products used in electrical motors, transformers, resistors, etc.

Mechanical assembly department—assembles components for airframe construction.

In addition to the personnel necessary to these departments, there is the usual executive and clerical staff needed to run a successful business.

The cases of disability among the 350 employees at Abilities, Inc., have been classified as follows:

A. Neurological conditions
   1. Cerebral palsy
   2. Dystonia
   3. Epilepsy
   4. Erb's palsy
   5. Multiple sclerosis
   6. Anterior poliomyelitis
   7. Cord injuries
   8. Parkinson's disease
   9. Guillain-Barré syndrome

B. Traumatic deformities (ranging from destroyed digits to bilateral amputation)

C. Peripheral vascular disease
   1. Buerger's disease
   2. Arteriosclerosis
   3. Hemiplegia

D. Heart disease
   1. Rheumatic
   2. Arteriosclerotic
   3. Hypertensive
   4. Congenital

E. Arthritic disease
   1. Rheumatoid arthritis (peripheral-spinal)
   2. Osteoarthritis

F. Auditory defects
   1. Congenital
   2. Acquired—otosclerosis, infection, trauma

G. Infectious disease
   1. Pulmonary tuberculosis
   2. Osteomyelitis
   3. Bronchitis, emphysema

H. Congenital deformities (ranging from extensive structural defects to mildly deformed extremities)

I. Speech defects
   1. Dumbness
   2. Severe stammering and stuttering

J. Visual defects
   1. Acquired—infection, trauma, metabolism
   2. Congenital

K. Allergic Disturbances
   Asthma (disabling with emphysema)

L. Geriatric Group (Many in this group have multiple disabilities which would place them in several of the above categories)

All of these disabled employees have been studied clinically, by means of a general history and physical examination and a laboratory routine that includes a complete blood count, urinalysis, and chest x-ray film. Additional procedures such as metabolic studies and electrocardiography are provided as indicated in particular cases. In every case, there is a detailed evaluation of the specific disability. The course of all these problems is followed by periodic examinations. The records of five employees are summarized below, to illustrate what is accomplished at this enterprise.

Case Histories

CASE 1.—A 39-year-old man became ill with rheumatic fever at the age of 12. He made an uneventful recovery following this initial attack and was well until he was 30 years old, at which time he developed shortness of breath with moderate exertion. This dyspnea progressively increased to the point where he was no longer able to work when he was 33 years old. In addition to his progressive dyspnea, he began to experience severe chest pains associated with cough and hemoptysis.

He was hospitalized at Meadowbrook Hospital, Long Island, on Dec. 1, 1956, at which time the diagnosis made was pulmonary infarction and rheumatic heart disease with enlarged heart, atrial fibrillation, mitral stenosis and insufficiency, and congestive heart failure, class 4E. A chest x-ray film made on Dec. 1 revealed a considerably enlarged heart involving all chambers and a pulmonary consolidation in the right lung field consistent with the diagnosis of pulmonary infarction. A repeat x-ray film, made Dec. 4, did not reveal any changes except for the possibility of a pericardial effusion. A third x-ray film on Dec. 7, showed massive cardiac enlargement, both right and left. Both chambers were still enlarged. There was marked enlargement of the left atrium, elevation of both main stem bronchi, and marked posterior displacement of the barium-filled esophagus. A fourth roentgenogram, dated Dec. 12, showed no further change except for some flattening of the right costophrenic sulcus. An electrocardiogram made on Dec. 1 showed the following: atrial fibrillation, prolonged intraventricular conduction, and right ventricular preponderance with possible hypertrophy. An electrocardiogram on Dec. 10 and 20, showed no change from the initial tracing.

Therapy at the hospital consisted of bedrest, intramuscular injections of mercurials, digitalis, and penicillin. After discharge from the hospital, the patient was unable to find employment because of his severe cardiac disease. He was hired at Abilities, Inc., in spite of his advanced rheumatic heart disease, and was given a job at a bench, which required several hours of slow walking daily. He was examined in October, 1957, after working for six months. During this period, the patient had no difficulty except for some moderate exertional dyspnea which was readily controlled by mercurials and digitalis. The cardiac findings were as follows:

The heart was enlarged to the sixth interspace and to the left anterior axillary line. A moderate diastolic thrill was felt in the fifth interspace and the midclavicular line. A slight precordial heave during systole was noted in the third interspace. There was accentuation of the first mitral sound, while the second sound was soft and barely audible. A grade I blowing, soft apical systolic murmur and grade III harsh diastolic murmur with radiation to the left axilla were heard. The pulmonic sound was normal, accentuated and louder than the aortic second sound. The rhythm was irregular, with a rate of 68 per minute. Examination of lungs showed the chest to be barrel-shaped, with hyperresonance on percussion over both lung fields. Breath sounds were distant and equal. There were persistent inspiratory and expiratory wheezes scattered throughout both lungs. The liver and spleen were not palpable; no dependent edema was present. The chest x-ray film revealed considerable cardiac enlargement in all diameters with some perihilar congestion. The diaphragms were flat; the costophrenic angles were clear. The ECG showed right ventricular hypertrophy, atrial fibrillation, and digitalis effect. The erythrocyte sedimentation rate was normal, and complete differential blood counts were normal.

The employee was at that time receiving digoxin, 0.50 mg daily; intramuscular injections of a mercurial, 2 cc a week; chlorothiazide (Diuril), 500 mg daily; and prophylactic penicillin, 200,000 units daily given orally. The employee has proved himself capable of doing his assignment. He is an able and intelligent worker and has received progressive promotions to the point where he is now employed in a supervisory capacity. He continues to have moderate exertional dyspnea which does not interfere with his work, and he has not lost any time because of his cardiac status since he was employed. He continues to receive digoxin, 0.25 to 0.5 mg daily, and 2 cc of the mercurial diuretic intramuscularly once or twice a week. With this regimen he has had no undue difficulty. The employee states that since he has been employed at Abilities, Inc., he can walk a greater distance than previously and suffers less breathlessness on effort.

CASE 2—A 62-year-old man was well until 1951, at which time he developed a coronary thrombosis with severe myocardial infarction. He was hospitalized for 31 days, during which he developed severe congestive failure which ultimately responded to therapy. Upon discharge from the hospital, he was confined to his home until April, 1956. During this period of home care, he was maintained on digitalis and received mercurial injections three times a week. He had orthopnea, marked exertional dyspnea, and a severe cough. He developed diabetes mellitus in 1953. Serial electrocardiograms revealed extensive heart damage, left ventricular hypertrophy, evidence of old myocardial infarction, digitalis effect, first degree heart block, and intraventricular conduction disturbance. Because of his history of severe cardiac disease, he was not able to find employment until April 16, 1956, at which time he was employed at Abilities, Inc., without receiving a preemployment physical examination. He was examined on Jan. 29, 1957, when he complained of mild exertion dyspnea and a slight cough. Physical examination revealed an elderly man who did not appear in any acute distress. There was no evidence of cyanosis. He was moderately pale. The heart was enlarged to the left sixth interspace at the midaxillary line. No thrill was palpable. The sounds were regular, and there were only occasional premature contractions. The heart rate was 80 per minute. The blood pressure was 140/100. There was a grade II apical systolic murmur which did not radiate. Examination of the lungs revealed dullness at the right base with diminished breath sounds; no rales were audible. The abdomen was pendulous; the liver was enlarged 4 fingerbreadths below the costal margin. No fluid wave or shifting dullness was evident. There was 3+ edema of both lower extremities from the foot to the knee. The patient was reexamined in November, 1957. At that time, he stated that his dyspnea was not so severe as at his previous examination. There was no change in the size of his heart, but there was a decrease in the number of extrasystoles. The liver was no longer enlarged; the edema which was apparent on his previous examination had greatly diminished to the point where he had only 1+ edema of the ankles. His diabetic status since the time of employment had been controlled by diet. The amount of digitoxin was reduced to 0.25 mg every other day. His mercurial injections were reduced from three times weekly to once a week.

This patient has shown a dramatic improvement since being employed. Previous to his employment at Abilities, Inc., he had been considered completely disabled and no longer able to work. This resulted in his remaining at home four and one-half years.

CASE 3—A 46-year-old man was well until Aug. 9, 1950, at which time, while working as a deep-sea diver, he was forced to ascend rapidly because of an emergency. Decompression facilities were not immediately available, and the procedure was carried out one week later. He developed severe caisson disease with resultant paralysis of both lower extremities. All sensations in the legs were absent for superficial tactile sensitivity of both thighs. He had loss of bladder and rectal control. The patient was hospitalized for two years. During that time, he was confined to a wheelchair. In February, 1952, he developed bladder stones and a urinary infection. Between August, 1950, and February, 1952, he did not have any formal rehabilitation. He became severely depressed and felt that his days of gainful employment were ended.

He was employed at Abilities, Inc., in August, 1952, as a methods engineer. Although previously confined to a wheelchair, he learned to walk with four-point gait and began to take active corrective exercises. In 1955, a urinary bladder infection developed which readily responded to therapy. In 1957, he was hospitalized, and an attempt was made to break up the spinal cord adhesions by intrathecal catheterization. Following this procedure, a Knight spinal brace and leg braces were advised to increase his crutch-walking ability. He then went through a series of specific exercises and as a result of this had some return of function of the tibialis muscles bilatrally. Peroneal muscles also showed some return of power as did the quadriceps. Bladder and rectal control was, and still is, absent.

Since his employment at Abilities, Inc., this employee has been increasingly active at his work. He has proven to be extremely intelligent and inventive to the point where he has been able to suggest modifications on existing machines to increase the effectiveness of those disabled personnel working on these machines.

CASE 4—A 40-year-old man was born with an absence of both arms and both legs. There was a stump with immature toes about 3 inches below the right knee joint but no knee on the left. His arm stumps are tapered so that he can grasp objects in the stumps with great facility. He walks on stumps enclosed in leather casings and is able to perform all activities of daily living. He attended primary and high school in the usual manner. After graduation from high school, he had several minor jobs such as selling newspapers. In 1953, surgical amputation of the toe vestiges was accomplished, and an attempt was made to fit him with artificial legs. After a lengthy trial, it was found that he could not wear these prostheses as it caused too much irritation of the stumps, and he was forced to discontinue wearing them. He was advised to use only his leather boots.

His first regular employment was at Abilities, Inc., in 1952. He has proved himself to be an extremely proficient worker in the packaging department and is now foreman of his section. During his period of employment here, his health has been excellent. He has lost no time because of illness except during the surgical restorative attempt.

The employee is a completely self-sufficient person. He can dress and feed himself and take care of all his needs. He drives his own car to and from work, after adaptions in the car had been made so that it could be operated by a quadruple amputee. He has proved himself to be both physically and mentally adjusted to his work.

CASE 5—A 29-year-old man had evidenced serious and increasing muscle incoordination since childhood. In the few years prior to his employment at Abilities, Inc., this incoordination increased to a considerable degree. His symptoms first began at age 11 years, with abnormal movements of his lower extremities and trunk. This progressed to the point where he was almost completely incapacitated because of flail-like movements of his arms and legs and an inability to balance himself. His private physician sent a letter to Abilities, Inc., at the time of his employment in 1955 in which the physician stated that this patient's "great problem is a lack of coordination, a condition which stems from his childhood and is caused by a severe emotional illness. He has been thoroughly investigated neurologically several times and his neuro-muscular system is organically normal." In spite of his obvious handicap, he was hired on a sitting job as a coil winder, which work he performed very well in spite of his severe neuromuscular disturbance.

This man was not considered employable prior to his being hired at Abilities, Inc. As a result of this, his muscular incoordination increased considerably because of emotional aggravation of his condition. This progressed to the point where he was rapidly deteriorating. After his employment at Abilities, Inc., his general condition and attitude improved, and he was a serious and capable worker. There was, however, progressive deterioration as regards his neurological disorder. Upon examination at Abilities, Inc., for the first time the diagnosis of dystonia was made. In September, 1957, this employee was sent for further evaluation to Dr. Irving S. Cooper, who corroborated the diagnosis of dystonia musculorum deformans. He stated that the prognosis, if the disease is allowed to take its course, is complete incapacitation leading to a state of chronic invalidism during which time the patient would probably be bedridden. In an effort to arrest this disease and attempt improvement, an operation called chemopallidalthalamectomy was advised. This consisted of injecting alcohol into the pallidalthalamic nucleus on the right side. Following this procedure, there was great improvement in the patient's condition. His coordination was much improved. Soon, however, his condition worsened but did not reach the level of incoordination that he had manifested prior to this procedure. It was felt that the worsening of his condition was due to deterioration of the untreated cerebral left hemisphere. Chemopallidalthalamectomy for the left side was done in December, 1959. Following this procedure, there was again pronounced improvement in the patient's coordination, which has continued to date. Following the two surgical procedures, the distinct improvement in his dystonia has reached the point where he evidences only slight muscular incoordination. His rehabilitation has been most gratifying.

Discussion

At present, there are in the United States approximately 28,000,000 disabled persons. There is no question but that this figure will increase every year. The increase of home and highway accidents is more than enough to balance a steadily decreasing industrial injury rate. Improved medical and surgical care preserves in some state of permanent disability a high proportion of the accident victims with injuries that used to be considered fatal. As a result of general advances of medicine, the life expectancy has increased, resulting in more and

more people's reaching the age of susceptibility to chronic and disabling disease.

Rehabilitation services have been keeping pace with the demands of a population that is increasing, growing older, and experiencing a greater number of accidents. The need is being recognized by the government and rehabilitation centers who are constantly expanding their services in research and training. Those persons in the field of physical medicine are continuing their search for ways to ameliorate residual disability and better serve the handicapped. Industry, however, because of archaic criteria as to physical ability to do work, is not taking full advantage of this source of manpower.

Since ancient times, society has been prejudiced as concerns the disabled man. He has had great difficulty in assuming a normal role in society. He has suffered from ostracism or been the recipient of charity. This attitude toward the handicapped persists to this day, and, as one result, the handicapped person has a great deal of difficulty in securing employment. Employment managers continue to resist employing the handicapped. The reasons they advance are many, such as the fear that workmen's compensation rates and liability insurance rates would increase, that handicapped workers are more accident-prone, that the handicapped cannot adapt to complex machinery, and that they do not produce as well as able-bodied employees.

The experience at Abilities, Inc., has been directly contrary to these ideas. As regards safety, in a period of seven years there have been only four compensable accidents, none of which was related to the person's disability. Absenteeism at Abilities, Inc., has been lower than the average in comparable industry. As regards production, the proof of the success at Abilities, Inc., is its record of increased annual production and sales, which is several millions of dollars.

Conclusion

From our studies relating to the employees of Abilities, Inc., all of whom are disabled, it is apparent to us that the handicapped can be successfully employed and that they can compete on a production level with the able-bodied employee. The increased employment of the disabled will tremendously increase the resources and economy of the United States. In order to accomplish this, industry should modify its concepts of employability. Negative job specifications leading to rejection should give way to positive job specifications emphasizing abilities.

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Fluorides: A Critical Review

IV. Response of Livestock and Poultry to Absorption of Inorganic Fluorides—Cont'd.

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Chronic Poisoning by Fluoride

THE ABNORMALITIES of livestock which are induced by the ingestion of excessive quantities of fluoride over extended periods of time (months to years) have been referred to as chronic fluorosis, a fluorotic state, fluorine toxicosis, fluoride toxicosis, chronic fluoride-induced toxicosis, darmous, or dermes (in Algeria), and Vayunoppulu or Vayupotlu (in the Indian districts of Kurnool and Nellore).

The appearance of disease among farm animals in association with their prolonged exposure to unusual quantities of fluoride, in the peculiar environment of certain geographic areas, and as the result of certain components of feed otherwise well suited to the profitable raising of stock, has resulted in an increased effort in recent decades to learn how to raise these animals successfully under such conditions of exposure. Attempts have been made in many reviews* to define the degrees of exposure and attendant factors compatible with the economic production of livestock.

A few experimental investigations, involving various species of animals,† have been conducted to compare the biological responses to different levels of fluoride in the diet under more or less commonly acceptable conditions of animal husbandry. These investigations have been of two types: (1) Abnormally high levels of fluoride (as NaF, rock phosphate, clay phosphate, etc.) were fed in adequate and, occasionally, inadequate diets, to evoke frankly recognizable signs and lesions of intoxication by fluoride; and (2) slightly to moderately elevated levels of fluoride were fed to determine the maximum levels that, in various species of farm animals, were compatible with economically satisfactory animal husbandry. The variations in procedure here included measuring the ability of certain minerals and other components added to the diet, and of certain livestock-management practices, either to forestall or to alleviate the damage that ingestion of excessive quantities of fluoride otherwise might induce.

Chronic fluorosis in farm animals has an insidious onset, develops slowly, and involves, primarily, gross and microscopic changes in bone and teeth.[68] Secondarily, there is progressive deterioration of the general health of the animal, dependent upon, or at least in association with, progressive changes in the skeleton and teeth. Under conditions specified subsequently, lameness develops, and the animal ceases to graze, because of the pain associated with movement, or through loss of appetite, or perhaps for the two reasons combined. Individual animals may become incapacitated and die. The frequency of occurrence and the severity of the lesions vary considerably in a herd or flock. These lesions and other signs of illness provide no more than presumptive evidence that the disease syndrome is the result of the absorption of fluoride.[67, 177] However, they comprise a fairly characteristic pattern of disease for the experienced observer.

The manner in which fluoride is incorporated into the structure of bones and teeth has been described in vitro and in vivo in tissues from laboratory animals.[117, 118, 129, 142] The fluoride ion forms hydroxyfluoroapatite by replacing principally hydroxyl and some bicarbonate ions in the hydroxyapatite.[13, 141]

Bones and teeth normally contain hydroxyfluoroapatite, as a result of the absorption of fluoride from the average daily diet. The continued absorption of large quantities of fluoride

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*References 2, 4, 31, 32, 47, 69, 75, 95, 113, 119, 130, 131, 132, 133, 135, 138, 149, 158, 168, 169, 180, 184, 202, 205.

†Dairy cattle: references 128, 160, 165, 173, 177, 185, 186, 187, 188, 189, 190. Beef cattle: references 97, 164. Sheep: references 81, 83, 84, 85, 97, 148, 150, 151, 152, 175. Chickens: references 71, 72, 78, 79, 89, 106, 108, 121, 157, 159. Swine: references 17, 18, 24, 30, 60, 105, 106, 107, 120. Goats: references 43, 44.

in the daily diet, and its incorporation in increased quantities into bone and teeth, reportedly, appears neither to change the x-ray diffraction pattern nor to develop crystals recognizably different from those of hydroxyapatite in unaffected bone.[2, 13, 109, 160] However, a decrease in the size of the crystal lattice was noted in the presence of increased fluoroapatite.[12] The hydroxyapatite of bone and teeth has a cell edge, *a* 9.41A, slightly more than that of fluoroapatite, *a* 9.37A.[13]

Classification systems have been used to record the occurrence and to grade the degree of grossly observable defects of the teeth of sheep and cattle, including those induced by the ingestion of abnormal quantities of fluoride (cattle,[80, 97, 177] sheep[97]).

Fluoride-induced changes in permanent teeth* are described generally in the following terms: mottling (including the terms chalkiness and staining); erosions; hypoplasia of the enamel (defective cellular development and incomplete mineralization); and abnormal wear (with selective abrasion, deformity and peridontal infection).[2, 22, 24, 152, 160] There is not such specificity in any one of these lesions as to justify an unequivocal diagnosis of chronic fluorosis in any particular animal. When they are induced by the absorption of fluoride, they develop only during the processes of formation and mineralization of the dentin and enamel of a specific tooth.

Permanent teeth exhibiting such lesions provide one of the earliest clinical signs of the absorption of abnormally large quantities of fluoride.[40, 194] Fluoride-induced lesions occurring in a fully erupted tooth do not retrogress, but remain, as long as the tooth persists, as evidence of the occurrence of absorption of excessive fluoride during a specific period in the development of that specific tooth. Once established, moderate to severe dental damage induced by excessive quantities of fluoride may even progress, because of continued stress upon the weakened and softened dental tissues. Such dental damage becomes important to the well-being of farm animals, when the affected teeth wear unevenly, or excessively, or break with undue ease, or become sensitive to cold water. Any of these sequelae may lead to a seriously curtailed intake of the diet.[2] Somewhat similar lesions may be expected to occur in incisor, premolar, and molar teeth that develop during the same period of excessive absorption of fluoride.[68] Once a permanent tooth is fully formed and erupted, no significant change in its architecture is induced by any level of fluoride intake.[2, 40, 68, 186, 191]

Deciduous teeth, because of their advanced state of development at birth, and because of the limited transfer of fluoride through the placenta from mother to fetus, generally show little or no fluoride-induced markings or defects.[2, 21, 40, 83, 97, 139] One of the earlier investigations, concerned with chronic fluorosis, recorded the observation that foals born on an affected farm had no dental changes in the deciduous incisors, which erupted about the 10th day after birth, but had distinct erosions on the second incisors, which erupted at about one month, and marked effects on the third incisors, which erupted at about six months. These effects noted in the horse may well be explained by the differences in the histogenesis and the time of eruption of their teeth[198] as compared with those of ruminants. Statements in textbooks concerning the time of eruption of incisor teeth should not be considered to be absolute in this matter. The time of eruption of the incisors may vary with breed,[54, 97] despite the failure of Brown and his associates to find significant differences in the chronology of dental development between the breeds or sexes of dairy and beef cattle.[26] There is insufficient evidence to establish that the normal timetable for dental eruption is changed by the absorption of fluoride.[83, 150, 152]

Wide variation has been noted in the degree of dental damage occurring among members of a herd or flock equally exposed to fluoride.[152, 201] Quite apart from the variability of the ingestion of fluoride among animals, there are several histogenetic variables which contribute to the irregularity of occurrence, the asymmetry, and the degree of severity of dental lesions. The normal stages of dental development occur cyclically and rhythmically, but irregularly, rather than gradually and continuously. Also, the effect of fluoride on the formative processes of the dentin and enamel is variable. Changes noted in pairs of teeth are more commonly bilateral but may be unilateral.[51, 97, 198] The preliminary mineralization of a developing tooth proceeds from the productive dentin outward, and then later through the enamel layer. The mineralization proceeds from the top of the crown toward the neck of the tooth, thus influencing the development of a horizontally disposed pattern of mottling induced by fluoride. The root forms shortly before the eruption of the tooth. Members of a pair of teeth may vary, one from the other, as much as a few months in the time of development and eruption.[97, 192]

*Photographs of fluoride-induced changes in teeth are to be found in the following references: cattle—2, 35, 47, 97, 140, 160, 165, 173, 177; sheep—2, 22, 83, 84, 85, 150, 152, 174, 197; swine—106, photomicrographs.

The dental lesions of fluorosis tend to occur in lesser degree in the first and second erupted pairs of incisors, and in greater degree in the third and fourth pairs. For example, the first and second incisors of cattle which, from calfhood, had access to water containing fluoride in the concentration of 4.5 ppm were less severely affected than the third and fourth incisors.[51]

The concentrations of fluoride in teeth appear to be related directly to the time of eruption during a sustained period of elevated absorption. The fluoride content of the permanent incisors of sheep exposed to an elevated intake of fluoride in water varied up to threefold in decreasing order in the first, second, and third pairs. The premolars and the third molar teeth had equal concentrations of fluoride, and about 1.5 times more than the second molars. Dentin generally contains a higher concentration than the enamel in the same tooth.[2, 29, 83]

Table IV—Dose of Fluoride Versus Reduction in the Consumption of Food

SOURCE OF FLUORIDE	NUMBER OF CATTLE	LENGTH OF TEST (YEARS)	CONCENTRATION OF F IN TOTAL DIET (PPM)	END OF TEST: APPROXIMATE AVG DAILY F INTAKE MG./KG. BODY WEIGHT	END OF TEST: SIGNIFICANT DECREASE IN FOOD INTAKE (YEARS)	END OF TEST: DIMINISHED GAIN IN WEIGHT	REF. NO
Barn fed; uncontaminated diet+NaF	3[a]	7	48	1.04	>2.5	0	128
	3[a]	7	58	1.27	>2.5	0	"
	3[a]	7	78	1.57	>1.5	slight	"
	3[a]	7	108	2.01	>1.5	slight	"
	3[b]	5	48	0.96	0	0	"
	3[b]	5	58	1.13	>3.5	0	"
Barn fed; contaminated hay	3[b]	7	44[c]	0.75	0	0	"
On pasture, contaminated pasturage	3[b]	5	46[c]	unmeasured	unmeasured	0	"
Barn fed, uncontaminated diet+NaF	2[e]	15 mos.	108	2.26	0	0	97
	2[e]	15 mos.	208	3.98	>6 mos followed by slight recovery	>5 mos	"
	2[e]	15 mos.	308[d]	4.12	feed concentrate: >3 mos. hay: >6 mos	>4 mos	"
	2[e]	15 mos.	607[d]	6.13		immediate	"
	2[e]	15 mos.	907[d]	6.25	refused feed in 3 days	immediate	"
	2[e]	15 mos.	1207[d]	8.98		immediate	"

(a) Yearling Hereford heifers, starting weights avg. 453 to 523 pounds per lot (3 animals each) were on dry feed and wintered in average to thin condition prior to start on test.

(b) Yearling Hereford heifers, starting weights avg. 621 to 694 pounds per lot (3 animals each) were on good pasture all summer prior to start of test.

(c) Fluoride concentration in total ration calculated during period when incisor teeth were being developed by cattle on test.

(d) Because of effect on appetite, the dose of NaF was then administered in capsules daily.

(e) Bred heifers at start of test.

Hobbs[97] and Garlick[67] have described developmental abnormalities, injuries, and disease commonly occurring in the teeth of cattle that were not exposed to elevated concentrations of fluoride in the diet and therefore were attributable to other factors (sheep[63]). These defects in incisor teeth have occurred so frequently as to be considered the expected background or range of dental defects against which to assess changes induced by fluoride.

The sustained ingestion of excessive quantities of soluble and insoluble forms of inorganic fluoride induces progressive, gross and microscopic changes in the skeleton, as well as in the teeth of all species of farm animals.* Clinically, bony outgrowths of periosteal bone (exostoses) and roughened areas (some palpable or visible, others not) appear on the surfaces of long bones, ribs and the rami of the mandible.

In cattle, exostoses occur commonly on the metacarpal and metatarsal bones (considered to be more frequent in the latter),[188] the mandible,[106] the ribs (particularly ribs 7 through 12),[55] and less commonly on other long bones, the pelvis, the frontal bone, the maxilla, and the scapula.[10, 97, 160, 177, 178]

Early experimentally induced skeletal changes in mature cattle, which were not palpable in the living animal, were described, after a thorough removal of soft overlying tissues, as a thin mantle of periosteal overgrowth on the surface of the metacarpus and metatarsus. This progressed in animals subjected to ingestion of larger quantities of fluoride to a roughened and thickened surface, especially at the distal ends of the bones.[35, 148, 165, 189]

The ribs of cattle appear generally to contain higher concentrations of fluoride than the frontal bone, in which the concentrations are higher than those in the metacarpus and the metatarsus.[156, 189] In sheep, the mandible and femur contain more than the tibia and the metatarsus less than the tibia.[83, 84, 85] It has been suggested that the differences in the concentration of fluoride in the various bones may be related to the difference in the rate of mineral turnover, the more porous bones of the skull, ribs and os coxae being richer in fluoride than the long, more compact bones of the legs.[13]

Certain gross changes are noted. The sur-

*References containing photographs of bony lesions in the following species are: cattle—10, 35, 160, 165, 177, 188; sheep—168; swine—106; goat—10.

face of the bone changes from a smooth, lustrous, and yellowish-colored appearance to one that is roughened, lusterless, and bluish-white to chalky-white. The smallest diameter increases, and the periosteum is thickened and adherent.[107, 168] Exostoses, bony ridges several millimeters long and ranging in size from that of a pea to that of a small egg, project 4 to 5 mm above the surface of surrounding bone. The lesions may be bilateral. The shafts of affected bones and the rami of the mandibles exhibit thickening and a general increase in diameter. The articular surfaces, in uncomplicated fluorosis, are unaffected grossly or microscopically. In extensively involved bones, exostoses, complicated by mineralization of muscular attachments, ligaments, and tendons, occur on the margins of articular areas.[188] These changes occasionally progress to ankylosis.[34, 125, 126, 160, 165] Deformity of the mandibles with exostoses has been noted in sheep, cattle and, rarely, in horses living in zones of phosphorite-containing soils.[196] The marrow cavity may appear to be narrowed, with thickening of the compacta. In swine, the size of the rami of the mandible increases because of the increase in size of the marrow cavity, rather than an increase in the thickness of the wall.[107] Spontaneous unilateral or bilateral fractures of the ribs[10, 34, 177, 179, 196, 200, 201] and coxae[2, 171] have occurred. Osteosclerosis, as well as osteomalacia, has been reported as occurring in some seriously affected animals under variable conditions of age and nutrition.[2, 83] X-ray changes in bones of cattle have been noted. An increase in the density of certain skeletal structures was the commoner, but abnormal porosity was also seen;[162] there were also periosteal feathering and thickening, increased trabeculation, thickening of the compacta, and narrowing of the marrow cavity. The greatest porosity was subperiosteal.[173, 177] Bilateral spontaneous fractures of the ribs were followed by incomplete union, increased density and trabeculation.[162, 173] X-ray changes in the bones of sheep also have been reported.[84, 85, 168]

Table V—Dental Lesions Associated with Exposure to Elevated Fluoride

Animal	Daily exposure to fluoride: Source of fluoride	F concentration in source (ppm)	mg F/kg body weight	Age at start of exposure	Dental lesions	References
CATTLE	F_2 as soluble fluoride in feed	≅15 to 25	avg. 0.4 to 0.6	———	nil to slightly moderate markings and stain; no abnormal wear	96, 97, 140, 173, 186
	F_2 as natural fluoride in drinking water	5	avg. ≅0.95	———	changes comparable with those associated with 15 to 25 ppm in feed, above	51, 164
		14.4	———	———	" " " " 60 percent of 35 cattle showed these lesions	201
	F_2 as soluble fluoride in feed	40	avg ≅0.88 to 1.26	———	medium to well marked; no abnormal wear	96, 186
		50	avg. ≅1.2 to 1.64 (peak 1.91)	———	more marked than at 40 ppm; no particularly abnormal wear	
	F_2 as natural fluoride in drinking water	16	———	———	changes comparable with 50 ppm in feed above	164
	F_2 as soluble fluoride in feed	est. 77 or more	avg ≅1.7 to 2.5	———	severe dental changes: marked mottling,(a) staining and abnormal wear in some animals	97, 140, 173
	F_2 as natural fluoride in drinking water	est. 18 or more	" "	———	" " " "	164, 201
CATTLE	F_2 as raw rock phosphate in feed	———	1.2 and 2.0	———	slight to moderate	56
		200 or more	≅3.5 to 4	———	severely marked	127, 160, 165, 194
	F_2 as Pasturage contaminated with atmospheric, fluoride-containing waste from industry	12 to 14	———	———	nil	97
		27 to 44	———	———	slight to moderate	
		40 to 60	———	———	marked to severely marked	
	F_2 as Pasturage contaminated with atmospheric, fluoride-containing waste from phosphate-producing industry	———	5	———	severely marked (exposure included tooth-forming period)	191
		———	5	———	nil (exposed after tooth-forming period)	
	F_2 as Na_3AlF_6 in feed	———	60 mg F per animal per day for 195 days	1 calf	moderate	24
SHEEP	F_2 as NaF in feed	———	15 mg F per animal per day for 71 days	1 lamb	moderate	24
	F_2 as CaF_2 in feed	———	340 mg F per animal	———	well marked	199
SHEEP	F_2 as raw rock phosphate in feed	———	7 mg F per animal for 3 years	exposed after 1 year old	nil	148
		———	60 mg F per animal for 3 years	" "	mild to moderate	
		———	120 mg F or more per animal for 3 yrs.	" "	severe	
		———	1.5	exposed as feeder yearlings	slight discoloration	175
		———	6.0	———	fracturing and erosion(a)	
	F_2 as NaF added to drinking water	5	winter: avg. 9 mg F per animal ≅0.29 mg F/kg summer: avg. 13 mg F per animal ≅0.37 mg F/kg for 3.5 years	exposed after 1 year old	nil to very slight mottling; no abrasion (molar teeth fluoride content ≅1300 ppm)	150
		7 or less	———	first exposed at 3 to 6 mos. for 2 yrs.	nil	199
		10	winter: avg. 18 mg F per animal ≅0.57 mg/kg summer: avg. 27 mg F per animal ≅0.71 mg/kg for 3.5 years	first exposed at 13 to 14 months (animals penned)	moderately severe; selective abrasion (molar teeth fluoride content ≅1900 ppm) (a)	8, 150
		20	winter: avg. 37 mg F per animal ≅1.10 mg/kg summer: avg. 53 mg F per animal ≅1.45 mg/kg for 3.5 years	"	severe (b) (molar teeth fluoride content ≅5100 ppm)	

(a) The exposure to levels of fluoride in this table can only induce changes in a particular tooth if the period of exposure includes the formative period of dentine and enamel. The sequellae (chipped, fractured, abnormally worn and abscessed teeth) are not dependant upon continued exposure to elevated fluoride. The sequellae, following the initial, fluoride-induced dental changes, are related to attrition imposed with time and by the character of the feed.

(b) The severe lesions caused some unthriftiness but did not cause economic loss in these affected, pen-fed sheep.

The microscopic changes* include increased and progressive resorption of bone and replacement by new bone starting at the periosteal surface. The periosteum thickens and penetrates between the columns of newly formed bone and cartilage. The surface of the bone beneath the periosteum becomes irregular and spiny. Subperiosteal areas exhibit new bony deposits in columns without cartilage and bone with cartilage scattered in a matrix of somewhat delicate looking connective tissue. Occasional areas of increased numbers of more closely laid Haversian canals are indicative of new bone formation. Some loss of normal bony structure, increased osteoid tissue, and porosity are seen. The marrow cavity shows resolving columns of poorly stained bone between the trabeculae of newly formed bone. There is focal fibrosis of the marrow without active hemopoiesis.

One investigation of chronic fluorosis in cattle, moved into and out of pastures contaminated by fluoride-bearing wastes from industry, provided evidence of the rapidity and reversibility of the formation and the resolution of bone. Removal of the animals from contaminated pasture to housing and uncontaminated feed resulted in an arrest of the fluoride-induced resorption, while mineralization of the newly formed bone proceeded. The periosteal proliferation underwent partial resorption. The alternation of exposure and removal from exposure resulted in the formation of several superimposed zones, indicating, in the manner of the rings of growth of trees, the frequency and extent of the effect. The zones of resorbing bone were separated by thin zones of regenerating bone. The resorbing layers of bone shrank and tended to become sclerotic. In older cattle, the porosis seemed less significant and the sclerosis greater.[34]

The breaking strength of long bones appeared to decrease in seriously affected sheep and swine. One report on cattle stated that the bones were normal or definitely strengthened,[160] but the occurrence of spontaneous costal fractures in seriously affected cattle is clinical evidence of the development of structural weakness in the ribs.* A group of investigators[2] compared the results of two tests commonly used by engineers to investigate the strength of materials, namely, the strength on bending and the strength on twisting to the breaking point, when applied to the bones of a few exposed and nonexposed sheep. They related the dimensions of each bone to these two measurements and obtained a value for the intrinsic quality of the materials composing the bones. They concluded that the bones of fluoride-affected sheep were bigger and heavier on the average; anteroposterior diameter increased at the midpoint in comparison to length; had shells of compact bone about 10% thicker, resulting in a relatively greater external diameter; were more flexible (two to three times as strong and equally as flexible as wood); had a slightly higher breaking strength on bending, and a

*Photomicrographs of histopathology of fluorosed bones are to be found in references 6, 21, 34, 38, 47, 106, and 177.

*The following references deal with measurements of quantitative change in some of the minerals, in the ash content, and in the physical properties of bones from livestock that have ingested excessive quantities of fluoride: 2, 30, 87, 106, 107, and 175.

Table V—Continued

Animal	Daily exposure to fluoride: Source of fluoride	F concentration in source (ppm)	mg F/kg body weight	Age at start of exposure	Dental lesions	Reference
SHEEP	F, as NaF added to drinking water	0.3	——	exposed from birth on pasture almost 7 years	nil (molar teeth fluoride content = 300 ppm)	152
		10.0	——	"	slight mottling and selective abrasion[a] (molar teeth fluoride content = 2100 ppm)	
		20.0	——	"	severe (molar teeth fluoride content = 2700 ppm)	
		10.0	——	first exposure at 2.5 to 3.5 yrs of age on pasture for 2 yrs.	nil (molar teeth fluoride content = <1000 ppm)	151
		20.0	——	"	" "	
		10.0	——	first exposure at 3 months alternating: 3 mos exposed, 3 not	slightly moderate, some weakening of cutting surfaces	85
		10.0	——	6 mos exposed, 6 not	moderately severe, some fractures[a]	
		10.0	——	6 mos exposed, 3 not	moderate, some wear	
	F, as natural fluoride in groundwater for drinking purposes	≅6.0	——	exposed from birth irregularly for 1 year	chipping and mottling	136
		≅12.0	——	"	severe; marked wear on central incisors[a]	
		12.0 at time of survey	calc.: 43 to 73 mg F per animal	exposed from birth for 6 years	20 percent of flock with badly worn and broken teeth[a]	174
	F, on pasturage contaminated with fluoride-containing atmospheric waste from industry	≅61.0	——	exposed from birth	100 percent of flock severely affected	21, 22
		≅44.0	——	"	~60 percent severely affected	
		≅16.0	——	"	high percent of flock with much less effect than at 44 ppm above	

Animal	Daily exposure to fluoride: Source of fluoride	F concentration in source (ppm)	mg F/kg body weight	Age at start of exposure	Dental lesions	Reference
SWINE	F, as NaF added to feed	290	——	exposed from weaning for 2 or more yrs. as reproducing females	moderate to severe	106
		580	——	"	" "	
	F, as NaF or Na_2SiF_6 added to feed	——	15 mg F per animal, for 117 days	exposed 8 weeks old	moderate	24
	F, as raw rock phosphate added to feed	330	——	exposed from weaning for 2 or more yrs. as reproducing females	moderate to severe	106
		650	——	"	" "	
		569	≅19 to 26	exposed as young pigs for 2 years	severe	60
		140	——	"	moderate	

20% lower breaking strength on twisting. These investigators point out that the strength of the whole bone is a product of size, shape, thickness of the shell, and the strength and quality of the material of which it is composed. They further observed, making calculations from data reported by Keck and associates,[106] that the strength of an individual bone as a whole (bending moment) remains high until a considerable amount of fluoride is present, whereas the strength on bending to the breaking point falls steadily with increasing content of fluoride, but without significant change in bone ash.[2, 106, 107, 108, 148] Thus, the tendency toward weakness in fluoride-affected bone is countered to some extent by an increase in the thickness of the shell of compact bone and an increase in the external diameter of the shaft.[87, 106, 160] Derangements in the metabolism of bones due to mineral imbalance in the diet, insufficient food through lack of appetite, or chronic illness may modify significantly the ash content of bones while the absorption of fluoride remains unchanged.

Lame, stiff, stilted, and painful locomotion may appear gradually or suddenly in animals affected by chronic intoxication with fluoride.* Slight, shifting lameness may, at its inception, involve one or more movements of one or more of the legs.[185] It may diminish, disappear, and recur at varying intervals in the same leg or movement or in another.[177] One opinion has been expressed, that rapidly induced skeletal changes in adult animals are more likely to cause lameness than those which take place gradually.[21] Lameness may occur with apparent abruptness, following the beginning of lactation in cattle that continue to ingest amounts of fluoride that are little less than those capable of inducing severe intoxication.[186] Not all of a flock of sheep or a herd of cattle or swine will exhibit lameness, even though their intake of fluoride would appear to be the same.[2, 177, 201] Lameness may become permanent in animals that continue to ingest excessive amounts of fluoride. These lame animals prefer to recline, and are reluctant to rise or move.[2, 10, 191] When forced to move, some cattle have been noted to propel themselves on their knees with the hindlimbs extended. They may stand with legs crossed or with weight-shifting movements.[68]

The lameness and painful gait commonly diminish and disappear[190, 191, 194, 200] in a matter of days or weeks, when the ingestion of fluoride by a seriously affected animal is terminated, only to reappear if the absorption of fluoride is resumed.[2] When extensive periarticular exostoses and ankyloses are present, lameness generally persists after the absorption of fluoride (in excessive quantities) has been terminated.

The concentration of fluoride in the food may be increased to the point at which nausea and unpalatability play a role in reducing the ingestion of the contaminated food.[97, 106, 108, 175] The greater the dose and concentration, the sooner the consumption falls off, as shown in Table IV.

An occasional episode of reduced intake or relatively abrupt refusal to eat may occur following the larger consumption of food (and of fluoride in contaminated food) which accompanies the start of lactation. The appetite may improve, with gain in weight, toward the end of the lactation period if the concentration of the fluoride is in the relatively low range.

Cattle, and more especially sheep, eat little or not at all when suffering from various severe degrees of irregular dental wear, malocclusion, alveolar periostitis, and peridontal abscess formation, all of which are sequelae of dental changes initially induced by excessive fluoride. These animals chew the usual feed and forage poorly and less willingly but appear to handle chaffed feed better.[83, 106, 179] Severely worn and abraded teeth may be sensitive to cold water, so that the affected animals lap at the water and slobber when attempting to drink.[2, 106, 160, 190] Such animals drink readily when offered water warmed to body temperature.[165]

The consumption of water by sheep and swine tends to increase with increase in the concentration of fluoride in their diets.[87, 106, 107] Sheep increased their voluntary intake of block salt while ingesting excessive fluoride as rock phosphate.[87]

Occasionally, the consistency of feces varied from normal with the continued ingestion of a diet containing elevated quantities of fluoride. This appears to occur unpredictably, and not frequently.[186] It is more likely to occur during an exacerbation of intoxication, or during a period of debility. Its occurrence has been reported among field cases[99, 191] and occasionally under experimental conditions (beef cattle[97, 168] fed on diets containing 308 ppm and more, $\cong$ 4.12 mg. of fluoride per kg., as NaF; swine[154]).

The quantities of fluoride retained in the body (that portion of the total quantity ingested that is not accounted for in the feces and urine) tend to be larger with increase in the dietary level of fluoride.[53, 97, 126, 185] The retention may range from about 12% at 30 to 40 ppm in the diet to 57% at about 100 ppm in the diet.

*References 2, 10, 21, 37, 40, 47, 57, 106, 122, 125, 126, 139, 164, 168, 186, and 200.

The possibility has been explored that elevated quantities of fluoride may induce changes in the gastroenteric flora.[97, 102, 103] The results of such experiments have been somewhat ambiguous.

It is common practice among livestock nutritionists to measure the apparent digestibility of nutrients (dry matter, crude fiber, crude protein, nitrogen-free extract, and ether extract) by measuring the proportion of the ingested materials, expressed in percent, which are evacuated in the feces. This provides some measure of the utilization of feed. The variations observed in measurements of this type were insignificant in cattle given feed containing fluoride in concentrations as high as 108 ppm[97, 185] and in sheep fed at levels as high as 208 ppm.[97] There is some evidence, among affected animals of a lowered conversion of feed to flesh (swine[60, 106]) and to butterfat (cattle[186]).

No significant change in the metabolism of calcium, inorganic phosphorus, or nitrogen in cattle or sheep could be attributed directly to the ingestion of elevated quantities of fluoride (cattle: 7 to 107 ppm, and 3 to 4 mg. of F/kg; sheep: 31 to 206 ppm).[97, 122, 123, 185]

Systemic evidences of chronic intoxication with fluoride appear only after local lesions have developed. The initial signs are likely to be inanition, loss of appetite, and painful locomotion. The general state of health and growth deteriorates, and weakness, emaciation, and cachexia develop later.

The prolonged ingestion of elevated quantities of fluoride has no direct effect upon conception, gestation, or parturition.[2, 165] However, serious dental damage and loss of appetite, with an attendant loss in weight and markedly reduced lactation, may result in the suppression or interruption of estrus,[60, 194] reduction of the weight of newborn, and poor subsequent development of young.[2, 10, 56, 60, 105, 106, 156, 200]

Laying hens raised and fed on a diet containing fluoride (as raw rock phosphate) in the

Table VI—Bony Lesions Associated with Exposure to Elevated Fluoride

Animal	Daily exposure to fluoride: Source of fluoride	F concentration in source (ppm)	mg F/kg body weight	Age at start of exposure	Duration of exposure before appearance of lesion	Type of lesion	Reference
CATTLE	F, as NaF added to the feed	30	avg. range 0.63 to 0.91	exposed as 2 yrs old for 5 lactations, dairy	5.5 years	thin mantle of periosteal overgrowth on bones carefully cleaned at slaughter	188,189
		30	"		3 years (no pasture)	" "	
		40	avg. range 0.88 to 1.26	exposed as 4 to 6 years old (2 to 4 lactations) for an additional 3 lactations, dairy	"	increasing roughness and thickening;[a] some mineralization of periarticular structures and ligamentous attachments on bones carefully cleaned at slaughter	
		50	avg. range 1.20 to 1.60		"		
		20	avg. range 0.40 to 0.61		"	nil	
		14 to 94	0.32 to 2.12	exposed as calves, 3 mos. old	372 days	rib biopsies gross and microscopic, nil	185
			~2.0	exposed as 2 years old, dairy	2 years	nil	173
			~2.5	"	"	slight exostoses	
		7		exposed as 20 to 24 mos. old, beef	422 days	nil	97
			~3.0 to 4.0	exposed as 3 to 5 yrs. old, hillcattle	within 8 to 10 months	exostoses	122,145
		107	≃2.2	exposed as 20 to 24 mos. old, beef	422 days	exostoses	97
	F, as NaF in a capsule, orally	≃907 in feed	≃6.25	" "	71 and 104 days, resp.	exostoses	97
		≃1207 in feed	~9.0	" "	40 and 81 days, resp.	exostoses	97

Animal	Daily exposure to fluoride: Source of fluoride	F concentration in source (ppm)	mg F/kg body weight	Age at start of exposure	Duration of exposure before appearance of lesion	Type of lesion	Reference
CATTLE	F, on pasturage contaminated with fluoride-containing atmospheric waste from industry	exposure uncalculated but elevated F in hay ~20 to 80 ppm F in Dust on hay ~260 to 480 ppm		various ages	after 2 to 3 mos.	palpable exostoses on ribs	139
	F, as raw rock phosphate in feed	~200 or more	400 ppm est as ~6mg/kg Ref. 4 857, 793	experimentally exp as 6 mos old, clinically, at various ages	~5 to 6 years	exostoses	40,47, 160,165
		———	calculated as 12 to 20	exposed as calves, and as lactating cows	periods up to 5¾ years	nil	56,140
	F, as natural fluoride in drinking water	25 to 32	———	———	most of lifetime	exostoses	201
		14.4 to 18.6	———	" "	" "	nil	
	F, as NaF added to drinking water	———	~4.91 and 5.35 mg F per animal	2 calves, starting wht 50 and 86 kg	10 months	exostoses	126
SHEEP	F, as NaF in feed	———	15.0	exposed as one 25 yrs old, one 6 mos old	1 month	exostoses	167
		206	———	exposed as 1 yr old	140 days	nil	97
		111	≃3.0	"	3 years	nil	
		112	≃3.9	exposed as weanlings	98 days	nil	81
	F, as raw rock phosphate in feed	———	120 mg F or less per animal	exposed as 1 yr old	3 years	nil	148
		———	160 to 170 mg F per animal	"	3 years	exostoses	

Animal	Daily exposure to fluoride: Source of fluoride	F concentration in source (ppm)	mg F/kg body weight	Age at start of exposure	Duration of exposure before appearance of lesion	Type of lesion	Reference
SHEEP	F, as raw rock phosphate in feed	———	up to 6.0	exposed as growing lambs	136 to 235 days	no exostoses mentioned	175
		———	" "	exposed as ewes	2.5 to 3 years	" "	
	F, as NaF added to drinking water	10 and 20	———	exposed as 13 to 14 mos old, fed in pens	3.5 years	no exostoses mentioned	150
		" "	———	exposed as 2.5 to 3.5 yrs old, grazing on pasture	2.6 months	" "	151
		" "	———	exposed as 13 to 14 mos old, grazing on pasture	6 years	" "	152
SWINE	F, as NaF in feed	290 or more	———	exposed as weanlings	within 144 days	exostoses	17,106
	F, as raw rock phosphate in feed	330 or more	———	"	within 144 days	exostoses	107
		293 or more	11 to 14 or more	exposed as young pigs	5 years	size, smoothness, texture and color of bones changed. No exostoses mentioned	60
		140	———	"	"	no gross changes of bones	
	F, as NaF, Na_2SiF_6, Na_3AlF_6 (mineral cryolite), or Na_3AlF_6 (synthetic cryolite)		15.0	"	168 days	palpable exostoses of mandible, and lesser degree of diaphyses	168
CHICKEN	F, as raw rock phosphate in feed	380 to 1000	———	exposed from hatching	52 weeks	bones very light to chalky white color, lacked normal luster. No exostoses mentioned	71
		680	———	exposed at 16 weeks of age	24 weeks	bones with almost bleached appearance no exostoses mentioned	

concentration of 530 ppm, produced as many eggs over a five-month period as did control hens. The fertility and hatchability were also unaffected.[72]

The transfer of fluoride from affected mothers through the placenta to fetuses or through milk to the sucklings was measurable but trivial and induced no recognized changes in the young of cattle and sheep.[59, 70, 83, 186] Analyses of bones from near-term fetuses whose mothers had been subjected to the ingestion of excessive quantities o ffluoride indicate that the deposition of fluoride in the fetal skeleton is relatively slight.[21, 97] The lactation of cows was not measurably affected until the reduction in their dietary intake had effected a decrease in their body weight.[105, 168]

Table VII—Lameness Associated with Exposure to Elevated Fluoride

Animal		Daily exposure to fluoride: Source of fluoride	F concentration in source (ppm)	mg F/kg body weight	Age at start of exposure	Duration of exposure before appearance of lesion	Depressed feed intake	Depressed weight gains or growth	References
CATTLE	beef	F, as NaF in feed	40 or more	avg ≅1.4	1 to 2 yrs.	2 yrs. or longer	yes	yes	96, 97
	dairy		40	1.4	freshening 2 yrs. old	5th lactation	yes	no	186
			50	1.7	" "	3rd lactation	yes	yes	
	dairy		50	1.9	2nd to 4th lactation	3 additional lactations	no	no	189
			100	≅2.12	3 months	16 months	no	no	185
	bull		—	3.0	'adult'	7 mos. or longer	not mentioned	yes	123
	dairy	F, as rock phosphate in feed	440 and 880	—	117 to 193 days	during 1st lactation	yes	yes	160
SHEEP	feeder lambs	F, as NaF in feed	206	—	8 to 12 months	140 days	yes	yes	97
			106	—	" "	"	no	no	
			111	—	" "	3 yrs.	no	no	
	lambs	F, as raw rock phosphate in feed	—	1.5	growing	136 to 235 days	no	no	175
			—	3.0	"	" "	no	slight	
			—	6.0	"	" "	yes	yes	
	ewes		—	1.5 to 3.0	yearling	2 yrs. and more	no	slight	
			—	6.0	"	" "	yes	yes	
SWINE	self feeder in dry lot	F, as raw rock phosphate in feed	217	—	young pig	2 yrs.	no mention	yes	60
			140	—	" "	"	"	no	
			293	≅11 to 14	" "	"	yes (sows during suckling)	yes	
	st in pasture		293	" "	" "	"	" "	no	
	st in dry lot		569	≅19 to 26	" "	"	no mention	yes	
	st in pasture		569	" "	" "	"	no mention	yes	

The skin has been reported as inelastic, dry, and coriaceous, and the hair as lusterless, in seriously affected animals, debilitated as the combined result of a poor quality of food and a reduced appetite.[10, 83]

The hooves of cattle, in particular, in field cases of chronic fluorosis, may occasionally grow abnormally and in unusual length.[47, 122, 123, 145] It has not been established that this abnormality is fluoride-induced, as it has not been reproduced experimentally.[186]

Shupe and his associates examined the soft tissues of 92 dairy cattle of varying ages, subsisting under natural field conditions at an elevated level of fluoride intake and showing various degrees of fluoride-induced intoxication. There were no gross or microscopic changes in the soft tissues of animals, the bones of which contained fluoride in concentrations less than 4,000 ppm. Some animals with bones containing more than 4,000 ppm had dubious fatty changes and focal calcifications in the stroma and tubules of the renal medulla.[164, 177]

Weanling pigs given fluoride (as raw rock phosphate) in concentrations of 160 ppm in their daily diet for 144 days, developed nephrosclerosis, whereas the kidneys of pigs given fluoride (as NaF) in their diets in concentrations as high as 970 ppm, and (as CaF_2) in concentrations of 910 ppm, were unaffected. These findings were verified in a repeated experiment.[106, 107] Pigs given fluoride at 569 ppm (19 to 26 mg of fluoride per kilogram of body weight, in raw rock phosphate) in the diet for one year, exhibited degeneration of the renal parenchyma and hemorrhages in the heart and stomach.[60]

Some dairy cattle in a group given fluoride (in raw rock phosphate) in concentrations of 220 to 880 ppm in the diet were found to have an overgrowth of newly formed connective tissue and hyalin degeneration in the liver, kidneys, adrenals, and heart, but no changes in the ovary or thyroid.[160] Chicks and pullets on a diet containing fluoride (as NaF) in concentrations ranging from 340 to 5,430 ppm suffered no change in the phosphatase activity of the renal tissue.[89]

Before any signs of illness or disease could be discerned, decreased hemoglobin and hematocrit values were noted in samples of blood taken at intervals from cattle maintained in one experiment, on a diet containing fluoride (as NaF) in concentrations of 300 and 600 ppm,[97] and in others in diets which provided about 3 mg of fluoride per kilogram daily[124] or somewhat more.[146] When there were 100 and 200 ppm in the diet, the blood values decreased slightly and then returned to normal by the end of 422 days of feeding.[97]

The levels of calcium, inorganic phosphorus, and magnesium remained within normal limits in the serum of the blood of adult cattle, sheep, and older chicks maintained on diets containing elevated quantities of fluoride.[53, 89, 97, 98, 124, 144, 145, 163, 175] Some evidence has been obtained of

Table VIII—Nutritional Signs Associated with Exposure to Elevated Fluoride

Animal	Daily exposure to fluoride: Source of fluoride	F concentration in source (ppm)	mg F/kg body weight	Age at start of exposure	Duration of exposure before appearance of lesion	Effect on locomotion	Reference
CATTLE	F, as NaF in feed	50	avg. 1.4 (peaks 2.0)	exp. from 2 yrs. of age, after first calving	at 5th lactation	lameness and stiffness appeared for the first time	186
		100 to 200	avg. 3.0 to 4.0	exposed as yearling cattle	within 8 to 16 months	lameness and stiffness	5, 97, 122, 126, 185
	F, as raw rock phosphate in feed	———	est. ~6.0	———	common occurrence by 4th year	lameness	40, 47
	F, as natural F in drinking water	18	≅ 2.0	exposed from birth	during lifetime	lameness	164
		25 to 32 or more (a)		———	" "	~20 percent of herd lame	113, 201
	F, on pasturage contaminated with fluoride - containing atmospheric waste from industry	20 to 80	———	exposed as young adults and adults	1 to 2 years	lameness	21, 139
		98	———	exposed as adult dairy cattle	about 1 month	lameness	2, 23
SWINE (b)	F, as raw rock phosphate in feed	650	———	exposed as weanlings	5 to 7 months	reluctance to move and stiffness	106
	F, as NaF in feed	580	———	"	144 days	" "	106
		1000	———	exposed as 120 to 150 days old	40 to 75 days	leg stiffness	37

(a) Lameness was not mentioned in affected sheep or goats (2, 13). Evidence is lacking to indicate that exposure to abnormally elevated quantities of fluoride induces lameness in horses or chickens.

(b) Swine and horses were unaffected although cattle were lame while on pasture contaminated with fluoride concentrations about 300 ppm on pasturage (atmospheric waste from industry, mostly rock phosphate) (120).

the occurrence of measurable but slight changes in the normal relationships of these substances.[89, 124, 175] Various deviations from normal values have been found in severe chronic intoxication, but concurrent reduction in the intake of dietary minerals by the animals may have affected the measurements.[97, 163]

Various changes in the activity of alkaline phosphatase in the serum of cattle have been reported,[21, 124, 160, 163] but recent investigations indicate that the continued absorption of fluoride at moderately elevated rates does not induce a significant change.[176] Phillips[155] reported that, in the absence of other diseases of the bone, such as rickets or osteomalacia, the alkaline phosphatase in plasma increased in rough proportion to the level of the fluoride intake of cattle that were being given fluoride in concentrations (in raw rock phosphate) up to 880 ppm in their diets. The increase appeared to parallel the severity of the gross signs of chronic fluorosis. This observation needs corroboration.

The rate of coagulation of the blood was lowered in baby chicks fed on rations containing fluoride (as NaF or in raw rock phosphate) in concentrations of 710 ppm or more for eight weeks. These chicks were, at the same time, seriously stunted and undernourished owing to fluoride-induced intoxication.[108]

Despite the inhibitory effects of fluoride on certain enzyme systems in vitro, there is no sound evidence that the ingestion of elevated quantities of fluoride by livestock induces a consistent or predictable change in the activity of any enzyme system in vivo.[137, 144]

Tables V, VI, VII, and VIII summarize the available information on the concentrations and dosages of fluoride ingested by farm animals, in association with the ages of the animals, the duration of their exposure, and the occurrence of dental and bony lesions, lameness, and signs of nutritional impairment. The data included in the tables clearly point to variations in the responses of animals to a given level of dosage, under the influence, no doubt, of a number of other factors. Because of these many variations, a condensation of the data to specific levels of dosage in apparent causal relationships to signs and lesions is likely to be highly misleading. An example of how one may be misled relates to an observed increase in milk and butterfat production concurrent with an increase in fluoride in the total diet sufficient to induce signs of intoxication. The increase in milk and butterfat production was due, not to the elevated fluoride, but to improved management practices undertaken at the same time.[140] Thus, it is necessary to know that an exposed group of animals is adequately cared for and free of chronic debilitating diseases induced by agents other than fluoride before one can evaluate the risk involved in the exposure to fluoride in any manner.

Since the signs and lesions of chronic intoxication develop gradually over many months or even years of excessive exposure, attempts have been made to determine the maximum concentration of fluoride which may safely be ingested by various species of farm animals for the length of their productive lives. A subcommittee of the National Research Council in 1955 made certain proposals, as set forth in Table IX, with respect to the safe ranges of concentrations of fluoride in the total rations of various species of farm animals.[158]

The evidence indicates that the average dose of 1 mg of fluoride per kilogram of body weight (as NaF or other equally soluble inorganic compound of fluorine) ingested daily by cattle during the productive period of life is not associated with any demonstrably injurious or abnormal effect.[140, 164, 173] This is as nearly equivalent as is possible (considering the sources of error indicated below) to having fluoride in the concentration of 30 ppm in the diet of dairy cattle,[187] or 47 ppm in that of beef cattle[188] (with only low concentrations of fluoride in the drinking water).

Table IX—Safe Levels of Fluoride in the Total Ration of Farm Animals as Proposed by the National Research Council (1955)

Species of animal	Fluoride as NaF or other soluble compound of fluoride (ppm)	Fluoride as contained in phosphatic lime, rock phosphate or other relatively insoluble compound of fluoride (ppm)
Dairy cattle	30- 50	60-100
Beef cattle	40- 50	65-100
Sheep	70-100	100-200
Swine	70-100	100-200
Chickens	150-300	300-400
Turkeys	300-400	.....

It is not uncommon for investigators to give a single average figure (covering a period of a year or more) as representing the daily dose of fluoride (per kilogram of body weight) taken by a group of animals that were being fed on a diet containing a known concentration of fluoride. Such calculations ignore seasonal variations in intake, as well as the increases which occur during the periods of rapid growth of young animals and during periods of lactation. The Subcommittee on Fluorosis Problems of the National Research Council calls attention to the fact that the dose of fluoride changes with the growth, the development, and the reproductive processes of farm animals.[161]

It is incorrect to assume that a constant concentration of fluoride in the total diet results in a constant intake of fluoride.[160] During an investigation over the period of five years, in which dairy cattle were fed on measured concentrations of fluoride (as NaF) in the diet, variations in the daily intake of fluoride were noted[187] to the extent indicated below:

Concentration of fluoride in diet (ppm)	Daily intake of fluoride (mg/kg)
20 (range of yearly averages, 4 cattle)	0.40-0.61: peaks 0.47-0.82
30 (range of yearly averages, 4 cattle)	0.63-0.91: peaks 0.85-1.14
40 (range of yearly averages, 4 cattle)	0.88-1.26: peaks 1.09-1.59
50 (range of yearly averages, 3 cattle)	1.20-1.60: peaks 1.55-1.91
50 (range of yearly averages, 4 cattle)	1.27-1.64: peaks 1.47-1.78

The average daily intake of fluoride, on the part of four groups of sheep, during the period of 3.5 years in which their drinking water contained fluoride at four abnormal levels of concentration, respectively, varied according to the season of the year and diminished as the experiment progressed, as indicated below. [It was noted also, during the latter part of the experiment, that the heaviest sheep in the group drank more water (by 55 per cent), and therefore took in more fluoride (by 35 per cent) than did the smaller ones.[150]]

Concentration of fluoride in in water (ppm)	Average daily intake of fluoride in water during various seasons of the year and at the beginning and end of experiment, of 3.5 years duration (mg/kg)			
	Winter months		Summer months	
	First year	Last year	First year	Last year
2.5	0.17	0.08	0.20	0.13
5	0.29	0.16	0.37	0.27
10	0.57	0.32	0.71	0.54
20	1.10	0.66	1.45	1.05

Merriman and his associates observed the following range of changes over a period of three years in the average yearly concentrations of fluoride (ppm) in samples of vegetation in a pasture contaminated by fluoride from the atmosphere.[128] Blakemore presented similar evidence.[21]

Variation in the yearly concentration of fluoride in the vegetation of the same pasture lands of the same herds over the period of 3 years

Herd	Number of fields	Range of variation (ppm)
A	3	1-44
B	8	3-18
C	7	4-10
D	4	11-24
F	4	6-16
G	8	1-13

These findings emphasize the fact that significant variations in the daily intake of fluoride occur among and within groups of animals at different times when the concentration of fluoride in their total diet is uniform, and also that the concentration of fluoride in the food is subject to wide variations under environmental conditions that might appear to be constant.

Diagnosis of Chronic Intoxication by Fluoride

The definitive diagnosis of chronic fluorosis in livestock and poultry is critically dependent upon the following evidence: (*a*) the presence, in some degree, of characteristic signs and lesions; (*b*) an unequivocal history of exposure, at the proper time in relation to signs of absorption (during the developmental period, in the case of teeth), to elevated quantities of fluoride in the environment or in the total diet, including food and water, for a sufficiently prolonged period of time; (*c*) reliably obtained analytical evidence that bony tissues and teeth, or urine, contain concentrations of fluoride within limits known to be associated with signs and lesions of chronic fluorosis.

Such evidence, collectively, is sufficient to establish the role of fluoride. The presence or absence of disease induced by other agents must be determined in all cases and interpreted as an associated, contributory or irrelevant factor. Some further comment on each of the three specific types of diagnostic evidence, as they are dealt with in the literature, may serve a useful purpose.

Characteristic Lesions of Chronic Fluorosis

Dental fluorosis is described clinically in terms of three surface changes that are induced only during the development of the enamel of permanent teeth—mottling, disturbed mineralization, and hypoplasia. One or more of these changes, although suggestive but not definitively diagnostic,* may be the first and only clinical evidence of a biological response to the absorption of elevated quantities of fluoride. However, in the absence of other signs, these dental defects are likely to go unnoticed. Each permanent tooth is a good criterion, among others, for determining the relative degree of exposure to fluoride to which the animal was subjected during the time of development of the enamel of that tooth.[68] The damage sustained by a permanent tooth in its developmental stage may be evident, even if the tooth erupts a full year after the excessive absorption of fluoride has been terminated.[84] Therefore it may be difficult to relate the time of the eruption of a particular tooth to the period of excessive absorption of fluoride. The termination of the abnormal rate of absorption of fluoride, on the other hand, does not bring about regression of dental lesions, nor does continued exposure to excessive fluoride cause any lesions to progress, once the enamel and dentin have been formed.[40]

The damage inflicted on the dental enamel by the absorption of fluoride during the developmental period may be obscured, and certainly complicated, by the wear and tear on the teeth of animals that graze upon coarse and dusty forage in semiarid pastures.[99]

Skeletal fluorosis varies greatly in its severity with a large number of factors. In general, the evidence indicates that the degree of abnormality in the bony structures of farm animals is greater as their forage is more highly contaminated with fluoride.[2] The species of the animals,[2, 10, 21] their age,[21, 161, 172] their general state of nutrition and well-being, and the duration of the period of excessive intake and absorption of fluoride are prominent factors in the severity of the skeletal lesions. The lesions, themselves, reach a stage in which they are readily palpable, and, after some experience in the examination of animals of various species, the examiner can employ their presence and their anatomical distribution as important features in the diagnostic procedure. Radiography is a useful adjunct to manual examination, but extensive experience with the radiographic abnormalities of the skeletons of animals, including those mediated by the absorption of fluoride, is essential in the avoidance of errors of interpretation.[83] The changes in the density of the bones vary from animal to animal, and with a variety of other factors, but in general, they bear a fairly direct relationship to the quantity of fluoride absorbed by the animals.†

The systemic signs of chronic intoxication by fluorides in animals are by no means specific, and, indeed, are often, if not usually, secondary to skeletal or dental lesions which contribute to the disability of the animal with respect to foraging for its food or grazing and chewing. A state of malnutrition results from a gross reduction in the consumption of food (whether due to lack of appetite or to painful locomotion or mastication), with loss of weight, dullness and disarray of the hair, and dryness and generally poor condition of the skin and the horns (if any) and hooves. The general appearance of the animals, in short, is not good. If young, their growth and development are retarded, and the milk production of brood and dairy animals, as well as the production of meat for the market, by other animals, is interfered with much or little, as the case may be.[2, 10, 40, 74, 99, 116, 191]

The history of the exposure of domestic animals to fluoride is likely to be influenced greatly by conditions which have existed for some time in a specific geographic region, whether such conditions are natural, as in areas long known to have excessive quantities of fluoride in the soil, or whether they are due to industrial or other factors which have contaminated soil and vegetation. The farmers in such areas will be familiar with the situation that exists (or has existed in the past) in their vicinity, and will have some concept, more or less valid, of the effects associated with the absorption of excessive quantities of fluoride by their domestic animals. Excepting as some entirely new and unrecognized source of exposure of animals to fluoride has been introduced into a farming or grazing area, therefore, it is not likely that the poor condition

*A series of four articles[65-68] entitled "The Teeth of the Ox in Clinical Diagnosis" provides highly useful information on this subject.

†References 2, 20, 21, 22, 42, 114, 140, 149, 164, 186, and 188.

or the poor performance of such animals will be brought to the attention of the veterinary practitioner or the investigator, without a well-defined preconception as to its cause. A historical background for a situation will not often be lacking, therefore, but it will require investigation and a fairly detailed elucidation and interpretation in its environmental and clinical aspects, especially in relation to the duration of any exposure to which the animals have been subjected.

Analytical Evidence of Fluorosis in Domestic Animals

An analytical survey of the environment of animals, involving determinations of the fluoride dispersed in both particulate and gaseous form in the air of specific areas, in association with the analytical examination of the soil and vegetation in relevant areas, may be required for the interpretation of clinical and analytical observations on animals. Such a survey, in order to establish the facts pertinent to a specific situation, whether to fulfill purposes of preventive medicine and hygiene or to meet the necessities of a medicolegal action, may have to be elaborate and prolonged. It may suffice, under other circumstances, merely to obtain a few representative data. In either case, a discussion of the methods employed in securing such data, and of the factual considerations involved in their interpretation, would go far beyond the intended scope of this review. In lieu of such discussion references to useful sources of information are cited.*

Determinations of fluoride in the urine of representative animals under appropriate conditions will often serve, as will no other procedure applicable to the intact, living animal, to establish the facts with reference to the current rate of their absorption of fluoride. Thus the collection and analysis of the urine by satisfactory methods may, because of its specificity, be a highly important aid in the diagnosis of disease.[40, 111] Inasmuch as the sampling of the urine, the analytical procedures, and the interpretation of the analytical results can be dealt with properly only by persons with adequate training and experience, students of the subject are referred to sources of information,[2, 21, 40, 52, 94, 97, 156, 173, 185, 186, 203] with the advice that they acquaint themselves thoroughly with these matters before undertaking any serious investigative or diagnostic work.

Determinations of fluoride in the bones of a domestic animal, or in representative bones of representative animals in a group, may be used judiciously for diagnostic purposes in the case of a specific animal, or for the purposes of appraising the status of a group of animals maintained under substantially the same environmental conditions. Here again, in order to make proper use of such information, an extensive background of knowledge is required. Careful consideration must be given to such matters as the expected quantities and the mode of distribution of fluoride within various parts of the skeleton[2, 97, 150, 188] of animals of varying age,[2, 21, 97] and species[21] in relation to the nature, severity and duration of the environmental exposure to fluoride, and the actual time of its occurrence (whether current or in the past) in association with existing lesions in the skeleton (and teeth), as well as the roentgenographic changes therein.†

In short, what is needed is a complete and profound knowledge of the metabolism of fluoride, as it is affected by the state of health or ill-health of the animals concerned, and by numerous nutritional factors which may exert an indirect effect upon the behavior of fluoride in the animal organism. The fact that the available information on these matters is not complete in all respects, provides yet another reason for cautious adherence to facts, rather than presumptions and fancies, in the consideration of the problems of domestic animals which derive from their absorption of fluoride.

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CENTRAL STATES SOCIETY OF INDUSTRIAL MEDICINE AND SURGERY

will hold its Annual Winter Meeting on

December 9, 1961

Knickerbocker Hotel, Chicago, Illinois

The program of scientific presentations will be as follows:

"Proper Sites for Hand Amputations and Incisions"
William B. Stromberg, Jr., M.D., Assistant Professor of Surgery, Northwestern University Medical School, Chicago

"Open Injuries of the Hand—Early Treatment and Later Reconstruction"
William H. Frackelton, M.D., Professor of Plastic Surgery and Head of Department, Marquette University School of Medicine, Milwaukee, Wis.

"Burns and Plastic Repairs of the Hands"
John L. Bell, M.D., Assistant Professor of Surgery, Northwestern University Medical School, Chicago

"Industrial Fractures of the Hand"
Robert D. Larsen, M.D., Clinical Instructor of Surgery, Wayne State University College of Medicine, Detroit

"Acute Hand Injuries"
Adrian E. Flatt, M.D., Associate Professor of Orthopedic Surgery, University of Iowa College of Medicine, Iowa City

"Vascular and Trophic Disorders of the Hand"
Ormand C. Julian, M.D., Professor of Surgery, University of Illinois School of Medicine, Chicago

Editorial

Let's Take Another Look

THE QUESTION as to whether coal dust by itself can cause disabling occupational lung disease has more than once been answered with a firm and final "no." But the question has a disconcerting way of returning for reconsideration in the light of new findings by investigators who will not let the subject alone.

On the basis of epidemiologic studies by Collis in England, and of animal experiments by Sayers at the U.S. Bureau of Mines and Gardner at Saranac Laboratory, many of us for years believed and taught that coal dust per se is a biologically inert material that induces no fibrosis and hence can cause no disabling pulmonary disease.

The occasional case of nodular fibrosis in the bituminous miner employed in the Appalachian coal fields has been dismissed as the result of unusual exposure to silica dust arising from the rock in which the coal veins lie. In some circles, such nodulation has been ascribed to the fine dust generated from sand which is dropped on rails to provide adequate traction for mine motor cars.

English investigators have published extensively over the past decade on pulmonary changes among miners employed in the coal pits of South Wales and Durham, with the conclusion that simple pneumoconiosis can result from the inhalation of coal dust alone. Small islands of particles are said to accumulate throughout the lung in the interalveolar stroma, and to give rise to a reticulin fibrosis. The surrounding alveoli break down, resulting in a focal emphysema which, in turn, can cause increasing breathlessness and cough. In some instances chronic infection, presumably tuberculosis, is superimposed, producing areas of massive fibrosis. The question which now plagues us is whether this disease process occurs among our own soft coal miners. Whatever our answer, it should be submitted only tentatively until we, in turn, have accomplished studies that match those of the English in scope and technical quality.

Two articles in our current issue report a recent study of this problem in the central Pennsylvania coal field. Research groups headed by Lieben and Baier accomplished historical and clinical screening of some 5,000 active and retired miners and industrial hygiene studies of 14 mines. Chest roentgenographic findings, reported in accordance with the "Classification of Radiographs of the Pneumoconioses," published by the International Labor Office (1958), revealed a substantial prevalence of pneumoconiosis. Cases with pronounced findings (those with coalescent lesions) occurred among 12% of employed miners and among 31% of retirees. The occurrence of the symptoms, shortness of breath and cough, correlated reasonably well with the extent of change in the chest roentgenogram; but reduction of the vital capacity measurement failed to show this association and, indeed, appeared to be more closely related to age. The industrial hygiene studies revealed wide variations in the concentration of airborne dust and in its silica content. Traction material for haulageway motors, though high in silica content, contributed very little to the over-all airborne silica dust.

That the Pennsylvania studies have defects is recognized by their authors. The smallness of the group of miners who came forward to be examined could have influenced the findings either for or against the prevalence of lung abnormality. It is difficult enough to estimate the dust exposure of a group of miners by means of a study that is ongoing and current; and when an estimation of past exposures is made from present findings, particularly in an industry which in the meantime has undergone major technological change, such estimation is more than a little risky. But enough has come to light as a result of the present studies to force us to take another look, and a hard one at that, at a problem which not so long ago was neatly wrapped up and resting on the shelf.

Book Reviews

HOW TO LICK EXECUTIVE STRESS AND STAY IN TOP EMOTIONAL AND PHYSICAL TRIM. Robert Collier Page, M.D. Price, $17.50. Pp. 238. Prentice-Hall, Inc., Englewood Cliffs, N.J., 1961.

MR. EXECUTIVE: KEEP WELL, LIVE LONGER. Peter J. Steincrohn, M.D. Price, $4.95. Pp. 341. Frederick Fell, Inc. 386 Park Ave., New York 16, N.Y., 1960.

By LEON J. WARSHAW, M.D.

One of the most successful forms of publishing endeavor in recent years has been the "how to . . . ," or self-help books. Of the endless variety in this category, those on health topics have probably been most popular. Some of these have been of considerable help to the industrial physician seeking health education materials to implement his attempts "to encourage personal health maintenance," one of the cardinal objectives of his occupational health program as defined by the A.M.A. Council on Occupational Health. Recently, two books have appeared that were aimed specifically at "executives," a segment of the personnel often difficult for the industrial physician to reach before signs and symptoms of disease appear.

The arrival of Dr. Page's book was heralded by announcements directed specifically to company presidents and other "top executives." Written by a very well known industrial physician and published by a firm famous for its handbooks on virtually every phase of corporate management, it offered great promise. Unfortunately, it fell far short of this reviewer's expectations.

There is little, by and large, to quarrel with in the content of this book or in its focus on such psychological factors as "executive stress," anxiety, tension, and interpersonal relationships on the job and in the home. The needs for proper nutrition, adequate exercise, and suitable relaxation are affirmed. The evils of alcoholism and obesity are portrayed; there is even a section on the "sexual and romantic role" of the executive.

The way it is written, however, leads one to wonder about the author's concept of the executive as a prototype. It is a grandiloquent harangue full of bathos and cant. Its style is reminiscent of the barnstorming revivalist who would raise his converts to heaven by the sheer vehemence and volume of his preaching. Unfortunately, executives are more likely to be irritated by this book than persuaded to follow good health habits.

The book by Dr. Steincrohn covers much the same ground as Dr. Page's book. It, too, is replete with lists of "nine ways to . . ." and "eight rules for . . ." that apparently put living and working on a truly business-like basis. Like Dr. Page, Dr. Steincrohn makes frequent use of catchwords and well-turned phrases to stir the imagination of his lay reader and help him remember the point being made.

Dr. Steincrohn's book, however, reflects his skill as a professional writer, polished by the preparation of some 12 previous books for the lay medical reader and his nationally syndicated newspaper column on health topics. It is written smoothly and persuasively, and the device of adding a summary of the "highlights" after each chapter will help the reader retain the point being made. Its price being less than one-third that of Dr. Page's book is another telling point in its favor.

No health book can supplant the individual counsel given by an articulate physician, based upon a careful medical examination and an understanding of how that particular executive reacts to his work and to his life at home. Frequently those who need such guidance most urgently are least receptive to it. Those who can be beguiled into reading health handbooks written as persuasively as the one by Dr. Steincrohn are more likely to follow pathways to good health.

Dr. Warshaw is Medical Director for Paramount Pictures, Inc., and United Artists Corporation. He is author of the book, "The Heart in Industry," New York, Paul B. Hoeber, Inc., 1960.

Questions

The questions published in this column are edited versions of requests for information that have been received during recent months. The answers have been prepared by one or more competent authorities in the particular area covered by the question. Readers may address questions to the Editor.

Question: *What is the present thinking regarding the role of rehabilitation centers in the treatment of "low back" injuries?*

Answer: Of all employees receiving compensation for low back injury, there are but a few who give a story of a bona-fide mechanical injury. In most employees "disabled" with low backache one can find a prominent psychogenic factor. It should be added that prolonged discomfort and disability of a real organic low backache at times may produce emotional changes even in a healthy person.

Prolonged disability can be prevented in most instances of "low back injury" by immediate investigation of the circumstances under which the symptoms developed and by early complete evaluation of the individual involved by the physician to determine why he has a backache. A definite decision should be made as to the compensability and the employee so advised; this demands courage. Concomitantly the employee should be receiving aggressive treatment for his backache and when emotional changes are present, support, understanding, and treatment of them.

If the above statements are accepted, it may be concluded that one must treat the whole patient, not just his back. A rehabilitation center is only complementary and secondary to supportive (at times psychiatric) treatment. One should not "pass the buck" to the rehabilitation center.

Abstracts of Current Literature

Dermatology

Stinging Insects—a Threat to Life. J. H. Shaffer.* J.A.M.A. 177:473 (Aug. 19) 1961. (*Division of Allergy, Henry Ford Hospital, Detroit, Mich.)

In 1959 Wylie graphically portrayed for the lay public his experience with a swarm of angry wasps. This led to a not uncommon chain of events. Frightened laymen sought expert advice; the experts, after some intensive experience, decided to report on the problem. The result in this instance is a competent and useful review, which is highlighted by a remarkable close-up series (4 times normal size) of a honeybee stinging, disembowelling itself, and leaving the stinger and venom sac. The author describes in detail an emergency kit furnished his patients. It includes tourniquets, tweezers, sublingual Isuprel tablets, and epinephrine for aerosol inhalation. In addition, the indications and value of desensitization are discussed at length. —W.L.E.

The Effects of Pretreatment with an Allergen in Active and Passive Sensitization. E. Skog.* Acta dermat.-venereol. 41:135 (2) 1961. (*Department of Dermatology, Karolinska Sjukhuset, Stockholm 60, Sweden)

Experimentally, contact sensitization can be prevented in any of the following ways: (1) immunological tolerance produced by exposure to the allergen in utero; (2) exposure to large doses of x-rays or radiomimetic drugs; (3) the interference phenomenon in which exposure to a very potent allergen prevents sensitization to weaker allergen; and (4) prior oral or systemic administration of the allergen.

The author has reexamined the fourth type of phenomenon. Guinea pigs were pretreated orally with 0.3 ml of 1% 2,4-dinitrochlorobenzene (DNCB) in olive oil each day for eight days. After a rest, the treatment was resumed for eight more days. Then, the animals were exposed intracutaneously to sensitizing doses of DNCB. Contact sensitization was markedly inhibited as compared to a control group pretreated with olive oil.

In other experiments, pretreatment did not prevent recipients from accepting passive transfer of contact sensitivity via parabiosis. This new finding suggests that inhibition occurs at the site of active formation of delayed "antibodies." From a practical standpoint, these various methods of inhibiting contact sensitization have, so far, all failed when applied to man. —W.L.E.

Chronic Urticaria Due to Mold Hypersensitivity. W. B. Shelley* and R. Florence. Arch. Dermat. 83:549 (April) 1961. (*University of Pennsylvania, School of Medicine, Philadelphia, Pa.)

Chronic urticaria frequently poses a frustrating diagnostic and therapeutic problem. Therefore, it is with some pride that Shelley and Florence relate how they solved the problem in a case of widespread chronic urticaria of five years' duration.

The initial diagnostic clue came with the finding of a severe, persistent urticarial skin-test reaction to Trichophyton. This sensitivity was passively transferred by Prausnitz-Küstner techniques. A Trichophyton rubrum infection of palms and soles was subsequently cleared with oral griseofulvin, but the attacks of urticaria persisted. Further skin testing revealed urticarial reactions to many molds, indicating a broad range of cross-sensitivity to fungi of many species.

Another significant observation was made: every time a venipuncture was made the patient developed urticaria at the site. Intradermal tests with various elements in his blood indicated that erythrocytes carried the urticariogenic substance. The authors suggest, but do not prove, that absorbed fungal elements coated the red blood cells to be released for antigen-antibody combination only when the cells diffused from vascular channels, as after minor skin trauma. This sort of coating has been described before for certain bacteria. The authors further suggest this mechanism may be operative in some cases of so-called "autosensitization," in which the patient may appear to be sensitive to his own cells or tissues. In reality, the cells are coated with an antigen derived from a drug or microorganism.

NOTE: This important paper should be read by (1) persons interested in speculative aspects of autosensitization phenomena and drug eruptions and (2) physicians concerned with practical management of patients with chronic urticaria. The discussion of diagnosis, source of exposure, and treatment of mold hypersensitivity is concise and informative. —W.L.E.

Clinical Toxicology

Recent Studies of Byssinosis in the Lancashire Cotton Industry. R. S. F. Schilling.* Med. Lavoro 51:754 (Dec.) 1960. (*Department of Occupational Health, London School of Hygiene and Tropical Medicine, London, England)

On the basis of a recent epidemiological survey of cotton mills in Lancashire, Schilling reports a high prevalence of cases of byssinosis. He observes that the processing of low-grade cotton, imported from the United States and Nigeria, gives rise to byssinosis with a greater frequency than similar processing of cotton of a finer quality.

A good correlation is observed between dust concentration greater than 250 mg per 100 cubic meters of air and the measured prevalence of byssinosis. Monday symptoms, however, do not result as a consequence of an especially high exposure during this day. On the contrary, ambient

values for dust appear lower on this day than during the workdays of the week. According to the author, a permissible level of 250 mg may be accepted, although only a value of 100 mg per 100 cubic meter is completely safe. It follows that, since such an absolutely safe value is unpractical and difficult to be obtained, workers exposed to levels of dustiness greater than 100 mg should receive special attention in order to remove those more susceptible to the effects of the dust.

By "partial correlation" analysis, the author shows that out of the three usual components of industrial cotton dust—minerals, cellulose, and protein—only the last is highly associated with the occurrence of byssinosis. Medium-sized particles, ranging from 7μ to 2 mm in diameter, appear to have the highest correlation with byssinosis, although the finer as well as the coarse particles are also responsible, but to a lower degree, for the occurrence of this disease. —M.C.B.

Experimental Studies on Byssionosis in Man. A. Bouhys.* Med. Lavoro 51:760 (Dec.) 1960. (*Laboratory of Clinical Physiology, University Hospital, Leyden, Netherlands)

The inhalation of cotton dust by human subjects, who had not been exposed to similar dust previously, resulted in a conspicuous disturbance of pulmonary ventilation, as suggested by a prolonged nitrogen washout clearance. By an open circuit method, Bouhuys measured the amount of ventilation required to reach a level of 2% nitrogen in the expired air while breathing pure oxygen.

The lung clearance index, measured as the ratio of this ventilation requirement to the F.R.C., well exceeded the upper limits of normal performance in the two subjects exposed to cotton dust. This effect appeared reversible, delayed in onset, and reduced by tachyphylaxis in repeated exposures. The whole process is probably connected with a histamine-releasing substance present in cotton dust and similar in effect to the compound known as 48/80. In support of this belief, the author implies that evidence has been gathered in regard to release of histamine in human lung tissue incubated with cotton dust. Bouhuys tends to exclude the opinion that histamine already present in raw cotton dust has any great significance in the pathogenesis of this bronchoconstrictive effect. He also excludes the assumption that an allergic process is involved to any significant extent in the mechanism of this disease. —M.C.B.

Prophylactic Treatment with Calcium Versenate in Workers Exposed to Lead. O. Bartolozzi* and N. Zurlo. Med. Lavoro 51:607 (Oct.) 1960. (*Clinica Del Lavoro, Milan, Italy)

In a plant for production of storage batteries, workers exposed to lead were subjected to a routine treatment with edathamil (Versenate) once a year or more frequently, in order to prevent lead accumulation in the organism. The treatment consisted of two periods of 10 days each, during which 2 gms. of edathamil calcium (Ca EDTA) were given orally. The cycle was concluded with the administration of cyanocobalamine, liver extract and iron, given as an over-all prophylactic measure, the iron being justified to replenish the possible chelating effect of the edathamil in regard to this metal. As an immediate consequence of this treatment, a conspicuous increase in urinary elimination of lead is commonly noticed, while the coproporphyrins elimination is not significantly modified. After this treatment was started, the morbidity occurring at this plant decreased both in terms of lead intoxication and in general morbidity and absenteeism.

NOTE: The prophylactic use of edathamil to prevent lead poisoning is condemned by most American industrial practitioners as a dangerous philosophy as well as being a hazardous clinical practice. —M.C.B.

Experimental Toxicology

Experimental Data for Determining the Maximum Permissible Concentration of Benzene Vapors in Living and Auxiliary Quarters and on the Decks of Oil Tankers. I. G. Dvoskin. Gig. Sanit. 25:18 1960. Air Pollut. Control A. Abstr. 6:3597 (April) 1961.

A special test was devised for chronic, dynamic 24-hour vapor exposures of animals to benzene in order to simulate the air of closed areas and decks of oil tankers. It was found that a concentration of 100 mg per cubic meter caused changes in the leukocyte count, in the unconditioned reflex time, and in the conditioned reflex activity. A concentration of 20 mg per cubic meter produced changes in the latter two. Concentrations of 4-6 mg per cubic meter altered the threshold value of conditioned reflex activity. The maximum permissible concentration of benzene vapor recommended for these conditions is therefore 5 mg per cubic meter. —R.E.E.

The Combined Effect of Repeated Viral Infection and Exposure to Carcinogenic Aerosols on Pulmonary Tumor Induction in C57 Mice. D. V. Wiseley,* P. Kotin, P. R. Fowler, and J. Trivedi. Am. A. Cancer Res. Proc. 3:278 (March) 1961. (*University of Southern California, Los Angeles)

Four groups of mice were exposed concurrently under conditions of strict isolation to (a) washed air; (b) carcinogenic inhalants; (c) washed air subjected to repeated or overlapping viral infections; and (d) carcinogenic inhalants and repeated viral infections. An aerosol of ozonized gasoline shown to be tumorigenic on inhalation for A strain and C57 black mice was used as the carcinogen. Significant differences in alveologenic tumor rates were observed. In addition, keratin-inducing peripheral epidermoid nests were in-

duced in mice of groups (c) and (d). The histogenesis of these lesions and their possible relationship to squamous cancer is discussed. —R.E.E.

The Biotransformation of Benzidine. II. Studies in Mouse and Man. L. J. Sciarini* and J. Meigs. Arch. Environ. Health 2:423 (April) 1961. (*Yale University, New Haven, Conn.)

After intraperitoneal injection of benzidine in mice, the following proportional distribution of urinary recoveries were obtained: benzidine 10%; monoacetyl benzidine 3.4%; diacetyl benzidine 2.6%; 3-OH ethereal sulfate of benzidine 29%; monoacetylated 3-OH ethereal sulfate and/or glucuronide of benzidine 25%; N-hydrogen sulfate and/or glucuronide 18%; 3-OH glucuronide of benzidine 12%. In workers, the following were recovered: benzidine 3.6-5.6%; monoacetyl benzidine 1.6-5.4%; diacetyl benzidine 5.1-10%; 3-OH benzidine from conjugates 89.7-78.5%. The first listed are values for August and the second for March. —R.E.E.

Enhanced Toxicity of Ozone-Hydrogen Peroxide Mixtures. O. J. Dobrogorski* and H. E. Stokinger. Am. Indust. Hyg. A. J. 22:21 (Feb.) 1961. (*U. S. Public Health Service, Cincinnati, Ohio)

In concurrent four-hour exposures, hydrogen peroxide in concentrations of 3.3 ppm and above increased the toxicity of normally noninjurious levels of ozone for rats, mice, and hamsters. A moderate degree of protection against otherwise lethal doses of hydrogen peroxide was obtained by a single prior exposure to it. A cross tolerance against the organic peroxide, cumene hydroperoxide, was afforded also by a prior exposure to hydrogen peroxide. A tolerance was also produced by a prior exposure to ozone for mixtures of O_3 and H_2O_2, despite the fact that such mixtures normally show enhanced toxicity. —R.E.E.

Biological and Chemical Studies of Tobacco Smoke Condensate. E. L. Wynder.* Am. A. Cancer Res. Proc. 3:280 (March) 1961. (*Sloan-Kettering Institute, New York)

Long-term biological studies have been established that the phenolic and acidic fractions of tobacco smoke condensate contain the major promoting substances. Short-term tests with subfractions of the phenolic fraction show most activity for the fraction containing the majority of the phenols. Promoting activity has been demonstrated for phenol with 7, 12-dimethylbenz(a)-anthracene and benzo(a)pyrene. Evidence is presented to suggest that the biological activity is largely the result of initiators, primarily polynuclear aromatic hydrocarbons and promotors, especially as present in the phenolic and acidic fractions. The content of initiators and promotors can be influenced by including additives at concentrations of 3%-5% in the cigarettes. Results of these studies in terms of chemical constituents and biological activity are presented. —R.E.E.

Noise and Hearing

Peculiarities of Noise-Induced Hearing Loss: A Study of the Hearing Loss of Engine-Room Personnel. J. van der Waal.* Ann. Otol. Rhin. & Laryng. 70:208 (March) 1961. (*Rijnsburgerweg 10, Leyden, Holland)

The hearing of some naval engine-room personnel was studied using a Van Dishoeck audiometer (one in which frequency is continuously variable, hence the shape of tonal gaps can be accurately determined). Of the 234 ears measured, 197 showed a tonal gap of 15 db or more (15 db HL re the European normal, which means from 5 to 15 db HL on American audiometers). The "median dip" (one that is representative in both severity and locus) was a very narrow tonal gap, having a maximum of about 20 db at 4500 cps but dropping to 0 at 3000 and 8000 cps. After correction for presbycusis, the data suggest that most of the loss is suffered by the end of the first year of exposure. No compensable losses were found; i.e., the losses were confined to the high-frequency region and did not involve the speech frequencies (500, 1000, and 2000 cps). The author points out that the temperature in a typical engine room is so high that neither plugs nor muffs can be worn steadily and, therefore, suggests that it would be better to reduce the noise levels in these compartments. —W.D.W.

The Determination of Hearing Loss. R. Carhart.* Veterans Administration, Department of Med. & Surg., Information Bulletin Audiology No. IB10-115, March 7, 1960. (*Northwestern University, Evanston, Ill.) (Available from the Superintendent of Documents, U. S. Government Printing Office, Washington 25, D. C., 15¢)

This publication outlines the procedures for measuring hearing losses in veterans. The nature and significance of auditory impairments are reviewed, and the general principles of both pure-tone and speech audiometry are outlined. The problem of interpretation of audiometric results and the evaluation of hearing impairment based on speech audiometry and based on pure-tone thresholds are discussed in detail. The special types of hearing losses encountered are outlined, and the modern tests for distinguishing nonorganic from organic hearing losses are summarized. The administration and interpretation of four tests employed within the Veterans Administration to isolate nonorganic auditory deficiencies, namely, galvanic skin resistance audiometry, the Doerfler-Stewart test, the delayed speech feedback technique, and the Stenger procedures, are described. —R.G.H.

Methodological Aspects of Auditory Threshold Measurements. J. F. Corso* and A. Cohen. J.

Exper. Psychol. 55:8 (Jan.) 1958. (*Pennsylvania State University, University Park, Pa.)

Two experiments dealing with methodological factors and the absolute threshold of hearing are reported. In the first experiment, audiometric tests were administered to 38 subjects by the method of limits on right and left ears at 9 frequencies from 250 to 8000 cps in each of three experimental sessions. Results indicated that the average intersubject variability was greater than the average intrasubject variability for the frequencies tested. Intrasubject variances over the course of the three tests were not significant, but nearly all intersubject sources of variance were highly significant. It was concluded that the precision of psychological experiments involving auditory threshold measurements may be increased by using repeated measures on the same subjects.

In the second experiment, the right ears of 10 subjects were tested 10 times by the method of limits at 125 cps in each of two testing sessions separated by 24 to 26 days. Results indicated that the mean threshold values decreased about 4 db from trial 1 to trial 10 in each session. It was concluded that the practice effects which accumulated over the first five trials may significantly decrease the absolute threshold for a 125 cps tone. This shift, however, is probably too small to be of any practical significance in clinical audiometry. —R.G.H.

Internal Medicine

Variant Form of Angina Pectoris—Previously Undelineated Syndrome. M. Prinzmetal,* A. Ekmekci, R. Kinnamer, J. K. Kwoczynski, H. Shubin, and H. Toyoshima. J.A.M.A. 174-1794 (Dec. 3) 1960. (*University of California School of Medicine, Los Angeles, Calif.)

The authors have elected to name this particular syndrome upon which they are reporting "A variant form of angina pectoris." They have seen more than 23 patients with this syndrome in two and one-half years. In this form of angina, the intracellular potassium and carbohydrate are decreased, while there is increased intracellular sodium; these findings are associated with ST segment elevation in the electrocardiogram. In classic angina, there are increased intracellular potassium and carbohydrate and decreased intracellular sodium; these findings are associated with ST depression.

The clinical features of this variant form of angina are as follows: There is typical pain at rest or with ordinary activity. Neither increased work of the heart nor emotional distress precipitates an attack. Often, the pain is cyclic and of a regular pattern. Ventricular arrhythmias are frequent at the height of the pain; the pain often recurs at the same time each 24 hours. Glyceryl trinitrate (nitroglycerin) results in dramatic, immediate relief.

The electrocardiogram, if taken during an attack, shows elevated ST segments; after the attack the tracing reverts to its appearance prior to the episode. On occasion, the R waves become taller during the attack in the same leads showing elevated ST segments. Exercise tests do not cause pain, and the electrocardiogram shows either no change or ST depression. A normal tracing in a mild episode does not exclude the diagnosis. Failure to take a second electrocardiogram after the attack may lead to a false diagnosis of infarction. Apparently, this syndrome occurs only when a large coronary vessel is involved, for example, the one supplying the diaphragmatic area of the heart or the anterior or high lateral area.

The authors conclude that this variant is a definite entity; they feel that this form is relatively common. An excellent table of differential diagnoses is incorporated. —S.H.S.

Bronchial Asthma in Adults. J. H. Shaffer,* L. L. DiLella, and J. A. Marvel. J.A.M.A. 174:1810 (Dec. 3) 1960. (*2799 W. Grand Boulevard, Detroit, Mich.)

The authors arbitrarily divide wheezing adults into three groups: (1) asthmatics from childhood (allergic); (2) those adults who have recently developed an allergy; and (3) those adults who wheeze from nonallergic factors.

Wheezing in the elderly person should be critically evaluated for conditions other than allergy as bronchitis, bronchiectasis, pulmonary fibrosis, cardiac disorders, and intrabronchial or extrabronchial tumors. It should be remembered that in young adults cystic fibrosis of the pancreas is being diagnosed with increasing frequency, these persons having survived to adolescence because of the use of antibiotics. Cystic fibrosis can be diagnosed by the following means: (1) increased sweat chloride level (more than 60 mEq); (2) absent trypsin in duodenal contents; (3) evidence of chronic pulmonary disease; and (4) positive family history of the disease.

The authors, in discussing treatment, confine their remarks to allergic bronchial asthma. The basic therapeutic approach is as follows: (1) relief of bronchospasm; (2) reduction of edema and evacuation of bronchial mucus; (3) control of the allergic factors; (4) treatment of intervening infection; and (5) improvement of general health. —S.H.S.

The Prognosis of Essential Hypertension Treated Conservatively. M. Sokolow* and D. Perloff. Circulation 23:697 (May) 1961. (*Department of Medicine, University of California School of Medicine, San Francisco, Calif.)

The cases of 439 patients with essential hypertension treated conservatively (dietary measures and sedation) were followed for a minimum of five years or until death. Initial blood pressure readings taken after 20 minutes rest in the supine position were classified as Grade I (150-200/90-

110), Grade II (200-250/110-130), or Grade III (higher than 250/130). Similar gradations from I to IV of fundal changes, ECG changes, and heart size were made, and notations of cardiac, cerebral, and renal status were recorded.

While increasing severity of changes in fundi, ECG, and heart size were associated with increased mortality as might be anticipated, the more significant findings in this study related mortality with the three gradations of initial resting blood pressure. Grade I blood pressure patients experienced a five-year mortality of 20%; Grade II patients showed a 42% mortality and Grade III patients a 68% mortality over the same period. This study emphasized the prognostic value of standardized initial blood pressure measurement and provides a framework against which long-term treatment of mild and moderate hypertension can be tested. —J.J.T.

Surgery

Tennis Elbow. R. S. Garden.* J. Bone & Joint surg. 43:100 (Feb.) 1961. (*Preston, England)

The author reviews the numerous suggestions as to the cause of tennis elbow. This condition is one of middle age and rarely affects tennis players. The diagnosis is based upon tenderness over the radiohumeral gap and pain during resisted extension of the wrist. It is a limiting ailment which seldom recurs after the symptoms subside. Approximately 75% of patients can be cured or relieved of symptoms by the injection of a cortisone medication into the area of joint tenderness. Garden believes that it is caused by a relative contracture of the extensor carpi radialis brevis. In severe cases in which relief with cortisone injection is not achieved, he does a Z-shaped lengthening of the tendon of this muscle just proximal to the point where the extensor pollicis brevis and abductor pollicis longus cross the tendon of the extensor carpi radialis brevis. The author reports relief of symptoms in 50 such cases with no residual loss of the power of wrist dorsiflexion or in the efficiency of wrist grip. —O.E.M.

Traumatic Rupture of the Normal Spleen. T. Dennehy,* T. A. Lamphier, W. Wickman, and R. Goldberg. Am. J. Surg. 102:58 (July) 1961. (*Marblehead, Mass.)

The statistical data from 83 cases of splenectomy for traumatic rupture of the spleen encountered in three urban hospitals between 1947 and 1952 are presented.

Contrary to the findings of many investigators, these authors found that over 50% of these patients were in the first two decades of life. Of the 44 patients in these two decades, 57% were below the age of 10 years. They also report finding an indirectly proportional relationship between age and incidence for every decade to the age of 80 years. In the cases analyzed in this study 94% were caused by blunt trauma. This group had a mortality rate of 11% compared with 40% mortality rate in those cases in which spleens were damaged by the penetrative injury of a bullet or stabbing instrument. A definite mode of injury was not recorded in 4 of the 83 cases. The authors are in agreement that these "spontaneous" cases are, in reality, the results of trivial or unrecognized injury occurring at some time previous to the rupture.

Although it is possible for a spleen to rupture without symptoms, general or localized pain in the left side of the abdomen in association with nausea and emesis are so common that they might be regarded as the "splenic rupture syndrome." In this series they found that generalized tenderness and spasm along with hypoperistalsis are the predominant signs. Apparently half of the patients with demonstrable signs showed generalized tenderness or spasm and over a third had hypoperistalsis. Pain occurring in the left shoulder (Kehr's sign) was present in only 4% of the patients. Leukocytosis and a declining hemogram were valuable laboratory findings in support of this diagnosis.

It is stressed that splenectomy continues to be the treatment for splenic rupture. In most cases operation was done within nine hours from the time of injury. No correlation was noted between mortalities and delay in surgery. The authors conclude that the signs and symptoms of a ruptured spleen are variable and that the diagnosis may be obscure immediately after the injury. For this reason importance must be attached to any one sign or symptom if early diagnosis is to be made and treatment started. —J.T.A.

Grease-Gun Injuries of the Hand. H. H. Stark,* J. N. Wilson, and J. H. Boyes. J. Bone & Joint Surg. 43-A:485 (June) 1961. (*Los Angeles, Calif.)

The authors report their experience in treating five patients with penetrating wounds of the hand caused by high-pressure injection of grease or oil; these injuries were encountered in the past six years. Such injuries probably occur more frequently than the cases reported in the literature would indicate.

The mechanism of this type of injury, briefly outlined, is a jet stream of oil or grease striking the hand or finger, piercing the skin, and filling the tissue spaces. Through a minute portal in the skin this material disseminates through the tissues, where it will not necessarily follow anatomical soft-tissue planes. Because of the seemingly trivial nature of the injury, the patient often continues to work. With the onset within a few hours of intense pain, numbness, and pallor, he will seek medical attention.

Grease-gun injuries should be treated as surgical emergencies. If they are not treated through properly placed surgical incisions, adequate decompression cannot be obtained. Unless the pressure arising from the sudden distention is relieved, ischemia will result, and if it is unrelieved

it will cause tissue necrosis. Emergency surgery is also advantageous in that tissue irritation due to grease or oil is lessened, and the wound is rid of contaminated material. The importance of thorough exploration of all involved parts in a bloodless field and the inadequacy of small incisions at the point of entry are stressed. As much grease and oil as possible are removed from the fingers and palm by means of a blunt curette and sharp incision. Fingers inadequately treated can be expected to develop a varying degree of fibrosis and scarring. —J.T.A.

Medical Care Administration

Man and His Changing Environment—Historical Perspective. G. Rosen.* Am. J. Pub. Health 51: 1013 (July) 1961. (*Columbia University School of Public Health and Administrative Medicine, New York, N. Y.)

The appalling inequalities in health conditions that exist in the world today are directly and intimately connected with the fundamental problems of wealth and poverty. Poverty and disease are linked through inadequate nutrition, housing, clothing, insanitary living conditions, and prevalent cultural values. These, in turn, are linked with low income and lack of education. Aid must be given to the underprivileged members of the international community in social and economic terms if they are to deal successfully with their health problems.

Excessive social costs, or social inefficiency and waste, exist to a substantial degree in our whole system of medical care. They are to be found in the provision of drugs, in the practice of medicine, and in programs for the indigent. Social change for better or worse is always the work of human hands. We have a professional and moral responsibility to work for a social and cultural environment in which human beings can lead a healthier and happier life. —A.B.

The H.S.P.A. Medical Plan. R. Y. Noda.* Plantation Health 26:16 (July) 1961. (*Oahu Sugar Company, Ltd., Hawaii)

The medical care plan has evolved from the necessity of a company's rendering medical care to its employees and dependents in an isolated rural community. The Waipahu Hospital was built in the early 1920's, but in 1955 management decided that maintaining a hospital was too expensive and began utilizing facilities of Wahiawa General Hospital.

Prior to 1946, there was no formal agreement between the workers and the company as to responsibility for medical care. In 1946, the company and bargaining unit entered into an agreement outlining the medical plan. The present plan was negotiated between the sugar industry and the ILWU. The coverage included medical care, medication, and hospitalization with exclusions such as refractions and glasses; rest cures; dental service; treatment for acute and chronic alcoholism, drug addiction, self-inflicted injuries; nursing or domicillary care; workmen's compensation cases; psychiatric cases except for diagnosis; etc. While the contract applies to the bargaining personnel, the coverage and limitations apply to the supervisory and nonbargaining personnel as well.

Care is administered by two physicians, four nurses, one x-ray and laboratory technician, two male orderlies, one secretary-cashier, one clerk, and two administrative office workers. The plan membership runs in the neighborhood of 3,000 and normally handles 26,000 to 28,000 visits annually.

The cost of operating the plan is largely borne by the company. The 1960 net cost to the company was $127,000, or a cost of $193.11 per employee per year, or a cost of $0.0638 per hour. Premiums paid by employees were as follows: single employees $1.65 per month, employee and wife $2.50 per month with $1.10 per month for each child, with a maximum of $6.00 per month.

The quality of medical care is good, and monthly mortality statistics for various reportable diseases is comparable to the statewide figures. —A.B.

The Role of the Voluntary Agency in the United States—Present and Future. R. H. Hamlin.* Bull. Nat. Tuberc. A. 47:4 (July) 1961. (*Rockefeller Foundation, New York)

The general functions and objectives of the voluntary agencies are as follows: (1) Determine new methods to improve the health and welfare of the American people. (2) Provide demonstrations of means by which these improvements may be made by both voluntary and government agencies. (3) Inform the general public and the professions. (4) Supplement government programs with personnel, facilities, and services. (5) Protect the public interest by working for effective government agencies. (6) Develop comprehensive, balanced community health and welfare programs. (7) Promote and maintain legislation in the public interest. (8) Support research into causes of disease, deprivation, inefficient organization, and inadequate service.

There is some question whether voluntary agencies are in a position at the present time to work toward the accomplishment of these objectives with the same effectiveness as they have in the past. The machinery of many voluntary agencies has become inefficient and self-centered. One difficulty with national voluntary agencies has been their inability to terminate, modify, or consolidate local affiliates when a better organizational structure with increased effectiveness would result. Affiliates too frequently have a desire for self-perpetuation.

If the National Tuberculosis Association is to continue to justify public support and independent fund-raising campaigns, it must maintain

good programs which fulfill a national purpose. Good programs in the national interest in a rapidly changing age depend upon good, efficient, modern organizational structure.

Voluntary agencies have been one of the finest instruments for private collective action by countless givers, volunteers, officers, and staff members. The American people are not questioning the need for voluntary agencies but their competence to carry out the roles assigned to them. —A.B.

Environmental Physiology

Appraisal of Heat Exposures in an Aluminum Plant. W. F. Lienhard,* R. S. McClintock, and J. P. Hughes. Proc. 13th Internat. Cong. Occup. Health, p. 879, 1960. (*Kaiser Aluminum and Chemical Corp., Chalmette, La.)

For better understanding of the physiologic effects of environmental heat on the industrial worker, studies were made of heart rate, rectal temperature, and sweat rate in 13 aluminum plant workers employed as skimmers, an occupation which in summer months combines exposures to radiant heat loads of high intensity emitting from molten aluminum at temperatures of 1,382°F with exposures to high ambient air temperature and humidity. Telemeter recording of the ECG of workmen during actual job performance provides an instantaneous and continuous index of circulatory strain. An electronic binary counter enables the observer to obtain a cumulative record of total heart rate.

When reflective shielding equipped with windows of infrared reflecting glass was installed between the worker and the source of radiant heat, all three indices of physiologic heat strain underwent dramatic reductions. Thus, by using the workman as a biologic metering system, the industrial physician can evaluate engineering and medical procedures designed to control environmental stresses. —D.M.

Radiological Health

Dose to the Eye from Radiographic Procedures. E. F. Focht,* G. R. Merriam, Jr., M. Barnes, and J. A. Evens. Radiology 76:459 (March) 1961. (*Department of Radiology, New York Hospital-Cornell Medical Center, New York)

The radiation doses delivered to the eye with standard diagnostic medical and dental technics were measured by use of a phantom head and an ionization chamber. None of the diagnostic procedures delivered significant radiation doses to the lens. The largest dose for a routine procedure with 2.5 mm Al (total) filtration was about 0.6 r for a single anteroposterior skull view. A full-mouth dental series of 14 films using 1.5 mm Al total and a 3½ inch field resulted in a 1.2 r dose to the lens. Compared to the 200 r required for a minimal cataractogenic dose for a single exposure, the diagnostic doses to the lens, even for repeated exposures, do not approach a cataractogenic dose. —G.L.V.

Intermittent Radiation and the Life Span of Mice. J. T. Duhig* and S. Warren. Arch. Path. 70:486 (Oct.) 1960. (*Laboratory of Pathology, Harvard Cancer Commission, Boston)

The effects of acute large doses of ionizing radiation on many species of animals are relatively well known. Less well studied are the effects of intermittent or chronic radiation. In this study, groups of mice were exposed to total doses of 500, 1,000, and 1,500 r of x-rays in weekly doses of 100 r. Four specific conditions were particularly studied: murine leukemia, lung tumor, severe infection, and nephrosclerosis. In the exposed groups, the lung tumors and severe infections occurred earlier in life than in the control group but not in significantly greater numbers. Nephrosclerosis not only appeared earlier but had an incidence up to eight times that in the control group. The leukemias could be divided into a thymic group and a nonthymic group. Radiation had little effect on the incidence of the latter type but a profound effect on the thymic leukemia, which was rare in the controls. —T.S.E.

Exact Dose X-Irradiation of Various Regions of the Head and Visual Sensations—X-Ray Location Method of Study of the Reactivity of the Central Nervous System. G. K. Gurtovoi* and Y. O. Burdianskaia. Biophysics 5:406 (March) 1961. (*Institute of Biological Physics, U.S.S.R. Academy of Sciences, Moscow)

The perception of a flash of light from x radiation, known as x-ray phosphene, was first studied by Roentgen in 1895. Early research on this phenomenon lacked good dosimetry or adequate preparation of the subject. The current study used human subjects who were dark-adapted for one hour. The doses (not dose rate) of 135 to 210 kilovolt x-rays were measured in milliroentgens (mr). Normal subjects perceived the light flash with a threshold of the order of 1 mr. Other studies have demonstrated that this perception is a direct effect on the retina. Subjects who have had one eye enucleated perceive the light flash when the x-ray dose is delivered to the blind side, but this perception can be shown to be due to scattered radiation to the normal retina. Completely blind subjects have no light perception, even at doses as high as 3 r. —T.S.E.

The Response of Swine After Exposure to the Gamma-Neutron Flux of a Nuclear Detonation. K. T. Woodward*, G. M. McDonnel, P. S. Harris, W. J. Kirkland, and J. N. Shively. Am. J. Roentgenol. 85:179 (Jan.) 1961. (*Department of Biophysics, Division of Nuclear Medicine Chemistry, Walter Reed Army Institute of Research, Washington 12, D. C.)

Two hundred sixty-four swine averaging 83

pounds were exposed to mixed gamma and neutron fields during the 1957 field tests of atomic weapons. The animals were subsequently studied for physical signs of illness, acute lethality, and median survival times. The gamma and neutron components of the dose were measured by chlorinated hydrocarbon dosimeter systems and fission and activation foils. Neutrons contributed approximately 45% of the rad dose. Individual doses ranged from 410 to 2,475 rads. The severity of the subsequent physical signs of illness could be correlated with the dose, and both hemorrhagic and gastrointestinal types of death were seen in the group. The $LD_{50/30}$ was 486 rads, which figure agrees well with findings in other studies on similar-sized animals with x and gamma radiation. At the median lethal dose, survival time was 14 to 15 days. —T.S.E.

Criticality Accident Application of the Oak Ridge National Laboratory Badge Dosimeter. E. D. Gupton,* D. M. Davis, and J. C. Hart. Health Physics 5:57 (April) 1961. (*Health Physics Division, Oak Ridge National Laboratory, Oak Ridge, Tenn.)

In addition to the security identification photograph, the new Oak Ridge National Laboratory (ORNL) badge contains gamma and neutron films, various filters for assessing the beta-gamma dose, indium foil for the quick segregation of those receiving a significant neutron dose, gold and sulfur for the measurement of thermal and fast neutron fluxes, respectively, and provision for chemical and silver phosphate glass dosimeters for high gamma doses. The ability to interpret high-level neutron exposures requires further knowledge of the spectrum which is supplied by permanently mounted area Threshold Detector Units (TDU). The TDU consists of gold, Pu^{239}, Np^{237}, U^{238}, and S^{32} inside a B^{10} and cadmium shield. Determination of fission and activation provides an estimate of the neutron spectrum in five energy regions. —T.S.E.

Industrial Hygiene Controls

Biological Effects of Magnetic Fields—Negative Results. J. E. Eiselein,* H. M. Boutell, and M. W. Biggs. Aerospace Med. 32:383 (May) 1961. (*Livermore Radiation Laboratory, University of California, Livermore, Calif.)

Some of the newer propulsion systems potentially useful in space vehicles develop strong magnetic fields. There is a need, therefore, to study the effects of exposure of humans to such fields. It is noted that, among physicists and accelerator operators in industries using atomic energy, " 'magnetic phosphene' or flash light" has been seen in cases where an operator's head has entered an undulating field. Attention is invited to the findings by certain investigators who reported retarded tumor growth, retardation of growth and alterations of white blood counts in mice exposed to strong magnetic fields. In the author's experiment, white mice were placed in magnetic fields ranging from 8,800 to 14,400 gauss. No significant effects on either the growth rate, white blood count, or the Erlich's ascites tumors in the experimental animals were demonstrated. —M.K.

Experiences with Phase Microscopy. M. Sheinbaum.* Am. Indust. Hyg. A. J. 21:518 (Dec.) 1960. (*80 Centre Street, New York 13, N. Y.)

Phase microscopy has been found useful in dust-counting particle size analysis and in quantitative analysis for silica. A procedure is described wherein an ordinary microscope furnished with a special condenser and objective is adapted for phase microscopy. The characteristics of air samples viewed directly in the medium for collection are described, together with the relative merits of using one or another of the commercially available membrane filters for collection of samples. The technique and the advantages of using styrene monomer in preference to other media is discussed in a detailed description of the method used for analysis of silica. Several techniques for separating dusts and mounting samples are reviewed critically. —M.K.

Evaluating the Hazards of Automobile Gasoline Additives. A. R. Jones,* R. S. Brief, B. E. Bennison, and W. T. Hausheer. Arch. Environ. Health, 2:635 (June) 1961. (*Esso Research and Engineering Co., Medical Research Division, P. O. Box 45, Linden, N. J.)

An assessment of the effects upon the public health as a result of the addition of a new metallo-organic compound to gasoline is discussed. The methodology described was applied in a situation where gasoline is produced and tested. The principles and the approach outlined, however, could prove useful in other types of industrial activity where a new product or process may raise an air pollution problem.

The authors' methodology included three separate phases, i.e., the experimental, the field trial, and the commercial. The first phase comprised a literature search, toxicity tests of the new additive, and an evaluation of the air environment in which equipment utilizing the fuel was operated. Clinical studies of employees involved were also included. In the field trial phase, air sampling programs were designed and carried out to determine changes in selected communities before and after the introduction of the additive to automobile fuels. The importance of obtaining the consultative service of public health officials is emphasized.

Technical reasons prevented the third or commercial phase from being fully activated. It is largely a combination of the first two and calls for repeated toxicological, environmental, and clinical studies on a broad scale over extended periods. —M.K.

Abstracts

Determination of Toxic Substances in the Atmosphere of Industrial Plants in Czechoslovakia. V. Vasák. Ann. Occup. Hyg. 3:122 (Feb.) 1961.

This brief report outlines the procedures adopted by the Regional Hygiene and Epidemiology Stations of the State Hygiene Inspectorate. An attempt has been made to standardize the sampling equipment and analytical techniques for use in laboratories throughout the country. In cases where it appears suitable, two analytical procedures are given, e.g., a colorimetric and a polarographic method. If both methods are available to the laboratory, the chemist may select between them. Although all hygiene stations in Czechoslovakia are equipped with direct indicating tubes for a preliminary determination of harmful contaminants, analytical methods must offer greater accuracy because they are often used to obtain technical data to be used by sanitation engineers in designing or evaluating ventilating equipments.

The author states that the standard analytical methods are to be published at the beginning of next year after approval by the Ministry of Health, and then he lists 38 specific chemicals or elements for which standard techniques have been or are being developed. —E.C.R.

Recommendations of The Symposium on Emergency Resuscitation

Sponsored by the Scandinavian Society of Anesthesiologists
Stavanger, Norway, August 21-25, 1961

1. The method of artificial respiration recommended in an emergency is full extension of the head and blowing through the mouth or nose.
2. The immediate start of resuscitation should be given priority over clearing the airway and draining water from the body of the victim.
3. The prone position should not be regarded as superior to the supine position either with regard to patency of the airways or with regard to drainage from the lungs.
4. First-aid workers of all categories, school-children and the general public should be taught mouth-to-mouth and mouth-to-nose resuscitation.
5. Organizations and individuals concerned with teaching life-saving should try to obtain training aids, which include dolls or manikins, airway models, films and film-strips, diagrams, and pictures.
6. The use of airways and other adjuncts should be taught only to medical personnel, nurses, and recognized life-savers.
7. External cardiac resuscitation should be taught and used only in conjunction with artificial ventilation, and for the present its use should be confined to medical personnel, nurses, and recognized life-savers.
8. The best way of disseminating the knowledge of artificial respiration would be its compulsory teaching to school-children.
9. A central information bureau should be set up in each country to collect information about incidents occurring outside hospitals in which methods of resuscitation have been applied.
10. The World Health Organization should be asked to act as an international information center to collect and classify information from national centers.

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About the Authors

► EUGENE P. PENDERGRASS, M.D., is co-author of the article in this issue, "Pneumoconiosis Study in Central Pennsylvania Coal Mines—Medical Phase." This is his latest product of many years of study of the roentgenology of occupational disease of the lung, resulting in a list of published articles and a textbook, "The Pneumoconiosis Problem, with Emphasis on the Role of the Radiologist" (Springfield, Ill., Charles C Thomas, Publisher, 1958).

Dr. Pendergrass did his undergraduate work at Wofford College, Spartanburg, S.C., and the University of North Carolina at Chapel Hill. He received the M.D. degree from the University of Pennsylvania School of Medicine, Philadelphia, Pa., in 1918. After an internship at the Hospital of the University of Pennsylvania, he began his lifelong career at that institution as an instructor in radiology. He became professor of radiology at Pennsylvania in 1936 and director of the department of radiology at the University hospital in 1939. He is also Director of the Donner Center for Radiology Hospital and Director of the Department of Radiology, Jeanes Hospital in Philadelphia. He is consultant radiologist to several other hospitals, and serves as consultant to a list of government agencies. He is a medalist of several medical societies, in recognition of prize exhibits and of special lectures. He is a member of Alpha Omega Alpha, honorary medical fraternity, and past president of the American College of Radiology (1948-1949); in 1959 he was awarded the honorary degree, Doctor of Science, by Wofford College.

► W. WAYNE MCBRIDE, M.D., has been with the Division of Occupational Health, Pennsylvania Department of Health, since 1958 as a public health research physician.

Dr. McBride is a graduate of the Jefferson Medical College, Philadelphia, Pa. He interned at Harrisburg Hospital, Harrisburg, Pa. Prior to joining the Pennsylvania Department of Health, Dr. McBride was in private practice in Harrisburg. He is a co-author of the article in this issue, "Pneumoconiosis Study in Central Pennsylvania Coal Mines—Medical Phase."

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► THEODORE PESSAR, M.D., is senior author of the article in this issue, "The Disabled in Industry—Experience at Abilities, Inc." He briefly attended universities in Germany and France and then enrolled at New York University, New York, N. Y., from which institution he received the B.S. degree in 1936. He received the M.D. degree from Long Island College of Medicine, Brooklyn, N. Y., in 1940. Thereafter, he served an internship and residencies at Kings County Hospital, Long Island College Hospital, Hospital for Joint Diseases, and Bronx Veterans Hospital in internal medicine with emphasis upon pulmonary diseases and cardiology. He is certified by the American Board of Internal Medicine. He practices as an internist and serves part time as Medical Director of Abilities, Inc., Albertson, N. Y.

► ROBERT R. YANOVER, M.D., is co-author of the article in this issue, "The Disabled in Industry—Experience at Abilities, Inc." He serves as Director of Surgery, Parsons Hospital, Flushing, N. Y. He is also Chairman of the Medical Advisory Board, Human Resources Foundation division of Abilities, Inc., Albertson, N. Y. He received the M.D. degree from Wayne University, Detroit, Mich., in 1920. His practice is limited to surgery.

Statement of Ownership

Statement required by the act of August 24, 1912, as amended by the acts of March 3, 1933, July 2, 1946, and June 11, 1960 (74 Stat. 208) showing the ownership, management, and circulation of Journal of Occupational Medicine, published monthly at Chicago, Illinois, for October 1, 1961.

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5. The average number of copies of each issue of this publication sold or distributed, through the mails or otherwise, to paid subscribers during the 12 months preceding the date shown above was: 4,614. (Signed) Clark D. Bridges, Business Manager.

Sworn to and subscribed before me this 13th day of October, 1961. (Signed) Mary A. Moses. (My commission expires October 22, 1962.)

People and Events

► RUPERT C. BURTAN, M.D., has been appointed Chief Physician at the Whiting Refinery of American Oil Company, Whiting, Ind. Before joining American Oil, DR. BURTAN was Plant Physician for the B. F. Goodrich Footwear and Flooring Company in Watertown, Mass.

DR. BURTAN received his M.D. degree from Columbia University College of Physicians and Surgeons in 1947. He served his internship at Lenox Hill Hospital in New York City, after which he joined the medical staff of the Lago Oil & Transport Company in Aruba, N.W.I. He remained there for three years and then went to Greenland, first as Staff Physician, and then as Medical Director for North Atlantic Constructors.

In 1954 DR. BURTAN returned to Columbia University and resumed his studies in the School of Public Health and Administrative Medicine. He received the master of public health degree in 1955 and the doctor of public health degree in 1956. Shortly thereafter he was called to active duty with the U.S. Public Health Service and assigned to the International Cooperation Administration, U.S. Operations Mission in Tegucigalpa, Honduras. In 1959 he joined the B. F. Goodrich Company.

DR. BURTAN is a member of the Industrial Medical Association and the American Medical Association.

► CHARLES C. BLACKWELL, JR., M.D., has been appointed Medical Director for American Brake Shoe Company in Chicago. He succeeds L. E. HAMLIN, M.D., who has retired.

DR. BLACKWELL has been in private practice in Evanston, Ill., since 1955. For the last three years he has also served part-time as Assistant Medical Director of the Washington National Insurance Company in Evanston.

DR. BLACKWELL received his bachelor of science degree from Northwestern University and his medical degree from the University of Illinois College of Medicine in 1947. He served his internship at Philadelphia General Hospital, spent one year specializing in internal medicine at St. Francis Hospital, Evanston, and two years as a resident in internal medicine at Hines Veterans Administration Hospital, Hines, Ill. From 1953 to 1955, DR. BLACKWELL served as a Captain in the Air Force, assigned as internist to the Chief of Medicine, 11th Air Division in Fairbanks, Alaska.

► LAWRENCE W. DIBERT, M.D., has been named Regional Medical Officer for the Pennsylvania Railroad's Pittsburgh Region. He will direct the activities of the company's medical officers and their staffs in western Pennsylvania, eastern Ohio and northern West Virginia.

DR. DIBERT was graduated from Shippensburg State Teachers College in Pennsylvania and received his medical degree from the University of Pittsburgh in 1945. He served his internship at Altoona Hospital, Altoona, Pa., and later became a staff member there. He joined the Pennsylvania Railroad in 1957 as Medical Officer and has served since then in the Pittsburgh area.

He is a member of the Industrial Medical Association, the American Medical Association and the American Academy of General Practice.

► HOMER L. SKINNER, JR., M.D., has joined the staff of the Carroll Medical Center, Carroll, Iowa. Since 1957 he has been engaged in the practice of general surgery at Staten Island, N.Y., and at the same time serving as Medical Director for the Nassau Smelting and Refining Company and the S.S. White Dental Manufacturing Company.

DR. SKINNER received the B.A. degree from Ohio University and the M.D. degree from Western Reserve University in 1952. He served his internship at University Hospitals, Cleveland, Ohio, and took his residency in general surgery at the U.S. Veterans Administration Hospital, Des Moines, Iowa. He is an abstracter for surgery literature for this journal.

► ALBERT O. RYAN, JR., M.D., has been appointed Assistant Medical Director for Olin Mathieson Chemical Corporation's operations at

The Occupational Health Institute's Certificate of Health Maintenance was recently awarded the Florida Power Corporation, the first Florida firm to have its medical department approved by OHI. Receiving the certificate is M. F. Bunnell, center, Vice-President in charge of industrial relations. Making the presentation is Terry F. Tanner, M.D., regional consultant for the OHI. At left is L. A. Newton, M.D., company Medical Director.

Pisgah Forest, N.C. For the last five years he has been in private practice in Hollywood, Fla. Previously he served as Director of the Polk County (Fla.) Health Department.

DR. RYAN served with the U.S. Army for three years during World War II, and later served for two years with the U.S. Navy Medical Corps. He received his pre-medical and medical education at the University of Cincinnati, obtaining the M.D. degree in 1947. He served his internship at the Cincinnati General Hospital, and his residency in medicine at Christ Hospital in Cincinnati.

►LT. COLONEL HENRY J. DONNELLY has been assigned as Chief, Preventive Medicine Research Branch, with the Army Medical Service Research and Development Command.

COL. DONNELLY was born in Portrush, N. Ireland, earned a Bachelor of Medicine, Bachelor of Surgery, and Bachelor of (Art of) Obstetrics from Queen's University, Belfast, in 1944; a degree in Public Health from the National University of Ireland in Dublin in 1946; and an M.D. degree from Queen's University in 1948.

After a senior residency in Chest Diseases at the J. N. Adam Hospital in Perrysburg, N.Y., he joined the Mahoning Sanatorium in Youngstown, Ohio, where he was Assistant Medical Director for two years.

In 1951 he entered the Army, and went to Korea as preventive medicine officer. He returned to the States in 1954 for the Advanced Course at the Medical Field Service School, Brooke Army Medical Center, and the Military Preventive Medicine Course at Walter Reed Army Institute of Research.

From 1958 to 1959, COL. DONNELLY was with the MAAG Mission to Taiwan as Preventive Medicine Advisor to The Surgeon General of the Chinese Army. He then returned to Washington as Preventive Medicine officer with the Headquarters, Military District of Washington, from where he transferred to the Army Surgeon General's Office.

COL. DONNELLY is a Fellow of the American College of Chest Physicians, American Public Health Association, and American College of Preventive Medicine, and is a member of the American Medical Association, National Tuberculosis Association, the Industrial Medical Association and the Royal College for the Promotion of Health (England).

Announcement

Applications are being solicited for Scientific Exhibits for the 1962 Industrial Health Conference which will be held at the Pick-Congress Hotel in Chicago. Exhibits will be shown April 10 to 12. Application blanks and information may be obtained from Allen P. Skoog, M.D., G. D. Searle and Company, P.O. Box 5110, Chicago 80, Ill.

► WILLIAM J. BURNS, Director of Exhibits for the American Industrial Health Conference, was honored by the American Society of Association Executives on September 3 at the Society's 42nd annual meeting held in Denver, Colo. MR. BURNS was one of a select group of 34 of the nation's leading association executives to receive the Chartered Association Executive Award which gives special recognition to qualified association executives who have acquired broad backgrounds in all phases of association management and met rigid requirements covering education, experience, training, and association achievement.

► DANIEL C. BRAUN, M.D., has been appointed Assistant Medical Director, divisions and associated subsidiaries, United States Steel Corporation. He will be associated with ROBERT B. O'CONNOR, M.D., Corporation Medical Director, at the headquarters office in Pittsburgh, Pa. For the last three years, DR. BRAUN has been Plant Medical Director for the Corporation's Homestead District Works in Munhall, Pa.

DR. BRAUN studied chemical engineering at Carnegie Institute of Technology and then transferred to the University of Pittsburgh School of Medicine where he obtained his M.D. degree in 1937. He served his internship at Mercy

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People and Events

Hospital, Pittsburgh. During the next 10 years he completed a number of postgraduate courses in industrial medicine, hygiene and toxicology at the University of Pittsburgh, Long Island College of Medicine and Columbia University.

From 1950 to 1957 DR. BRAUN was Medical Director of the Industrial Hygiene Foundation of America and a Senior Fellow at the Mellon Institute of Industrial Research in Pittsburgh. Prior to that he was Medical Director for Pittsburgh Coal Company, serving subsequently as a consultant in occupational medicine for Pittsburgh Coal, Westinghouse Electric Corporation and other industrial firms in the Pittsburgh area.

DR. BRAUN is a former director of the Industrial Medical Association and is currently President of the Industrial Medical Association of the Pittsburgh-Cleveland Area. He is certified in occupational medicine by the American Board of Preventive Medicine and is a Fellow of the American College of Chest Physicians, the American Academy of Occupational Medicine, the American Public Health Association, and the Pittsburgh Academy of Medicine. He is also a lecturer in occupational health at the Graduate School of Public Health, University of Pittsburgh.

Certification

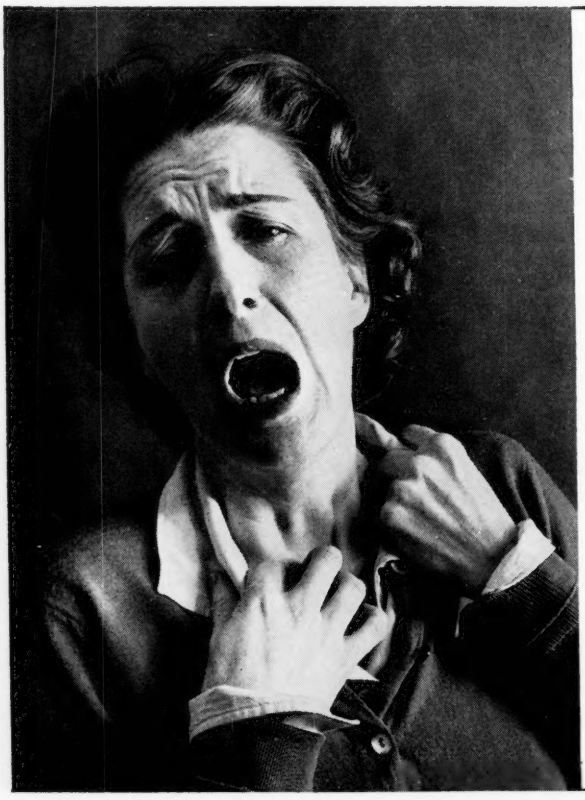
The American Board of Preventive Medicine announces that the next examination for certification in Occupational Medicine will be held at the Pick-Congress Hotel, Chicago, Ill., on April 7, 8 and 9, 1962.

Other changes in the medical division of U.S. Steel are as follows:

DAVID JOE SMITH, M.D., has been appointed to succeed DR. BRAUN as Plant Medical Director for the Homestead District Works. For the past two years, DR. SMITH has been Plant Medical Director at the Corporation's Edgar Thomson Works, Braddock, Pa. Before joining U.S. Steel, he was Medical Director for L. S. Ayres & Company, Indianapolis, Ind., from 1953 to 1959. He graduated from Indiana University Medical School in 1942 and interned at the Methodist Hospital in Indianapolis.

JOSEPH B. BENTZ, M.D., has been appointed to succeed DR. SMITH as Plant Medical Director at the Edgar Thomson Works. For the past year DR. BENTZ has been Plant Physician at Homestead Steel Works. He received his M.D. degree from Temple University Medical School in 1949 and interned at the York Hospital, York, Pa. Following two years with the army of occupation in Germany, he engaged in private and industrial medical practice in York for five years. In 1958 he obtained a master's degree in public health from the University of Pittsburgh's Graduate School of Public Health. This was followed by additional training in occupational medicine.

WILLIAM M. WOODWARD, M.D., has been appointed to succeed DR. BENTZ as Plant Physician at Homestead Steel Works. Since



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*Glaser, J.: Ann. Allergy 18:510 (May) 1960.

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1955, DR. WOODARD has been Regional Medical Officer for the Pittsburgh region of the Pennsylvania Railroad Company, following two years as internist for the railroad in Philadelphia. He received his M.D. degree from Jefferson Medical College in 1946 and served an internship at the Cooper Hospital, Camden, N.J. He spent two years as a captain in the U.S. Army Medical Corps before completing his residency training in internal medicine.

Calendar of Meetings

November 9-11, 1961—Hawaii Association of Plantation Physicians, Kauai Surf Hotel, Kauai, Hawaii. Executive Secretary: Miss Grace S. Withington, 1133 Punchbowl St., Honolulu, Hawaii.

November 13-17, 1961—American Public Health Association, Cobo Hall, Detroit, Mich. Executive Director: Dr. Berwyn F. Mattison, 1790 Broadway, New York 19, N.Y.

November 15-16, 1961—American College of Preventive Medicine, Detroit. Secretary: John J. Wright, M.D., P.O. Box 1267, Chapel Hill, N.C.

December 6, 1961—New York State Society of Industrial Medicine annual meeting, New York University Club, New York City. Executive Secretary: R. MacDiarmid, M.D., Room 2400, 30 Rockefeller Plaza, New York 20, N. Y.

December 9, 1961—Central States Society of Industrial Medicine and Surgery, Knickerbocker Hotel, Chicago, Ill. Secretary: A. P. Skoog, M.D., 55 East Washington St., Chicago 2, Ill.

February 7-9, 1962—American Academy of Occupational Medicine, Pittsburgh-Hilton Hotel, Pittsburgh, Pa. Secretary: Paul J. Whitaker, M.D., Allis-Chalmers Mfg. Co., P.O. Box 512, Milwaukee 1, Wis.

March 30, 1962—Michigan Industrial Medical Association, Parke, Davis & Company Research Laboratories, Ann Arbor, Mich. Secretary: Robert D. McIntosh, M.D., 6307 West Fort St., Detroit, Mich.

April 10-12, 1962—Industrial Medical Association, Pick-Congress Hotel, Chicago, Ill. Managing Director: Clark D. Bridges, 55 East Washington St., Chicago 2, Ill.

May 13-17, 1962—American Industrial Hygiene Association, Sheraton-Park Hotel, Washington, D. C. Executive Secretary: George D. Clayton, 14125 Prevost St., Detroit 27, Mich.

► Four physician appointments in the Headquarters Medical Department of Humble Oil and Refining Company have been announced by V. C. BAIRD, M.D., Medical Director. They are as follows:

ROBERT A. WISE, M.D., has been appointed Associate Medical Director and will coordinate medical services for the Southwest Region. He was formerly Assistant Chief Physician in the Medical Department of the Southwest Region.

H. B. SNYDER, M.D., has been appointed Associate Medical Director and will coordinate medical services for the Manufacturing Division and the Humble Pipe Line Company. He was formerly Senior Physician in the Medical Department of Standard Oil Company (New Jersey) in New York City.

H. T. ENGELHARDT, M.D., has been appointed Chief Internist and will be administratively responsible for the Clinic. He was formerly Internist in the Medical Department of the Southwest Region.

C. H. MONTGOMERY, M.D., has been appointed Internist and will coordinate Medical Services for the Marine Division. He was formerly Internist in the Medical Department of the Southwest Region.

► JOHN ALLEN, M.D., will discuss the role of company medical services in rehabilitation at a postgraduate course on "Rehabilitation of Patients with Injuries of the Spinal Cord" to be presented by the University of Wisconsin School of Medicine on November 16-18 at the Wisconsin Center. DR. ALLEN is with the Wisconsin State Department of Public Welfare and is the Industrial Medical Association's District Counselor for Wisconsin.

► Hawaii Association of Plantation Physicians will hold its 18th annual meeting at the Kauai Surf Hotel on the island of Kauai, November 9, 10 and 11. Roundtable discussions will be held on the subjects of Traumatic Surgery, Orthopedics, Pediatrics, and Dermatology. In addition, the following scientific papers will be presented: "Relief of Pain" by JOHN DILLON, M.D., University of California, Los Angeles; "Diagnosis and Treatment by Use of Radio-Isotopes" by R. NORDYKE, M.D., Honolulu; "Alcoholism in Industry" by ALOYSIUS S. CHURCH, M.D., Detroit; and "Changes in the Plantation Medical Plan" by CLINT CHILDS, Lihue, Kauai. A business meeting and election of officers will conclude the sessions.

► The first workshop session of a Medical Self-Help Training Program was held in Brooklyn, N.Y. on October 17. The new program, which is aimed at training 60 million Americans how to survive a national emergency as well as meet their own health needs if deprived of a physician's services, was introduced by the U.S. Public Health Service in cooperation with the American Medical Association. The program is to be implemented by a series of formal training lessons which will be augumented by the use of a reference manual to be provided as a resource document.

DAVID M. BENFORD, M.D., who addressed the session as a representative of the A.M.A.'s Committee on Disaster Medical Care of the Council on National Security urged physicians and members of all the health professions to provide the professional leadership and guidance that is so necessary for the successful teaching of the general population.

Similar workshops will be held in November and December in Alameda, Calif., and Battle Creek, Mich.

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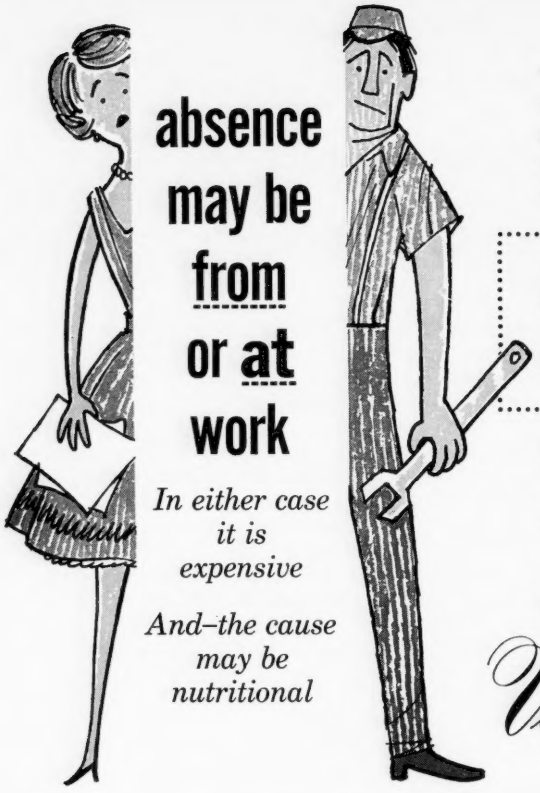

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